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A honeybee collecting nectar from an apple blossom. One of the first insects to be incriminated by experimental evidence as vectors of a plant pathogen. "The blight bacteria grow in the nectar and multiply there are saprophytes and then enter the tissues. The *Bacillus* of blight in the nectar is carried from flower to flower by insects visiting the flowers for pollen and honey. An artificial epidemic of pear blight started by infecting a few trees on the edge of an orchard and allowing free access of insects. Mosquito net bags over the flowers kept out insects, therefore protect the flowers from blight." (Waite, 1891.)

INSECT TRANSMISSION OF PLANT DISEASES

BY

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PREFACE

During the past twenty-five or thirty years, there has appeared in the literature of plant pathology a constantly increasing amount of evidence that insects play an important role in the spread and development of many plant diseases. Much of this evidence has never been adequately summarized or coordinated and is to be found only in widely scattered publications. In this book, the author has endeavored to bring together in one publication the most important contributions in this field of study. An effort has been made to evaluate and interpret the evidence in the light of the more recent advances in entomology and plant pathology.

Because the author feels that the subject has not received sufficient attention from teachers and research workers, he has presented it in a manner that frankly strives to emphasize the importance and promise of the subject as a field of study and research. The book is published, therefore, with the hope that it will stimulate greater interest in the subject among those students who are concerned with the problems of plant protection.

In presenting the material, an effort has been made to be brief. No attempt has been made to discuss all diseases transmitted by insects or all insects that transmit diseases. Emphasis has been placed on those which have been investigated most completely or which are best suited for illustrating the various principles of insect transmission. Moreover, in discussing a disease only those facts relevant to insect transmission have been included. Much information of general interest in plant pathology has been omitted purposely. In most cases, however, suitable references to such information have been given. In like manner, no complete technical descriptions of the insect vectors have been included. This information can be obtained readily from standard entomological texts or from the special references cited.

Because of the method of presentation, in which certain chapters deal with specific examples of insect transmission and

others with the principles involved, a small amount of repetition has been unavoidable. It is believed, however, that the added convenience to readers with different backgrounds of information will more than compensate for any objectionable features of this method of arrangement.

The author first became impressed with the potentialities of this field of study in 1923 when an investigation of the blackleg disease of potatoes led him into a study of the role of Dipterous insects in the spread and development of the bacterial soft rot of plants. Since that time, he has actively prosecuted research in some aspect of the association of insects with plant diseases. From 1932 to 1938, he taught a course in the subject in cooperation with Prof. A. A. Granovsky of the Department of Entomology and Economic Zoology at the University of Minnesota. A similar course is now taught by the author at West Virginia University.

The idea of writing such a book was originally suggested in 1929 by the late Dr. Royal N. Chapman, whose interest and enthusiasm caused the author to continue his activity in this field of study. Without Dr. Chapman's constant encouragement, the book would not have been written. The preparation of the manuscript was begun while the author was professor of plant pathology at the University of Minnesota and has been completed at West Virginia University. The author is indebted to his colleagues at both institutions for aid in many different ways. He is grateful to Dean E. M. Freeman for much helpful advice and encouragement. Thanks are expressed to Drs. Clyde Christensen, Carl J. Eide, E. C. Stakman, Leon H. Leonian, and Carlton F. Taylor for critically reading the entire manuscript and to Dr. Clarence A. Mickel for reading Chap. XIII and for aid in determining the correct names for a number of insects. Many helpful suggestions have been received from these colleagues, but the author assumes full responsibility for any errors in the text. Acknowledgment is due also to Dr. A. A. Granovsky, who was associated with the author in the course offered at the University of Minnesota, for reading the first draft of Chaps. I to VI.

The author is grateful indeed to those who have kindly furnished illustrative material, specific acknowledgment of which is given elsewhere. The illustrations not otherwise designated

are from the files of the Department of Plant Pathology of the University of Minnesota or the Department of Plant Pathology and Bacteriology, West Virginia University.

The references cited, with the exception of about 30 titles, have been checked for accuracy with the card catalogues of the Library of the U. S. Department of Agriculture or with originals deposited there. Grateful appreciation is expressed to the Bureau of Plant Industry Library staff for making these facilities available.

JULIAN GILBERT LEACH.

MORGANTOWN, W. VA.

June, 1940.

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INSECT TRANSMISSION OF PLANT DISEASES

CHAPTER I

INTRODUCTION

Since the dawn of civilization, man has been interested in the health of plants. In all probability, plants were subject to disease long before they were brought under cultivation, but the diseases were of little concern to man until he began to give special care to those plants that furnished him food. At first the diseases were attributed to supernatural phenomena, and their control was sought only through appeasement of the appropriate gods. With the development of more intensive agriculture through the ages, the necessity of protecting crop plants against disease has become progressively more acute, and consequently a more scientific attitude toward the problem has developed.

For many centuries, however, the study of plant diseases was dominated by dogma and superstition, for the nature of diseases remained obscure and little or nothing was known of microorganisms. In later years, when microorganisms were observed to be associated with diseased plants, they were interpreted, at first, as products rather than as causes of the abnormal conditions. It was not until the middle of the nineteenth century, when the classical studies of De Bary definitely established the pathogenic nature of certain fungi, that the subject was placed upon a true scientific basis. With this new concept of the nature of plant diseases, progress was relatively rapid and much was learned about the diseases of plants and how to control them. Naturally, however, emphasis was placed on the etiological aspect of the diseases, and for many years the more important works were descriptive in nature. Thought was dominated almost completely by the mycological viewpoint. This influence is still

very strong, and, even now, there are some who look upon phytopathology as nothing more than applied mycology. Much of the research in contemporary plant pathology is still descriptive and exploratory. Only in very recent years has attention been given to other and more fundamental aspects of the subject.

Even control measures, to a large extent, have been based on empirical studies with only a superficial understanding of the many factors involved. Spray programs and seed treatment practices, for the most part, are based upon incomplete data obtained empirically, and relatively little is known of the nature of the action of the fungicides or the factors that influence their effectiveness. Satisfactory control of plant diseases cannot be achieved until we know much more than we do now about their nature and the factors that influence their development.

A Neglected Borderline Field.—There is no aspect of the study of plant pathology more fundamentally important than that dealing with the methods by which diseases are transmitted and the relative significance of each method. Successful control measures, with few exceptions, are dependent upon a knowledge of these facts. Yet the subject has received only casual treatment, and the facts of transmission are known thoroughly for only a relatively small number of diseases. The role of insects in the spread and development of plant diseases has been especially slow in receiving the attention that it deserves. Plant pathologists have been inclined either to avoid a study of insects associated with diseases or to treat the problem in a very superficial manner. This attitude has resulted in the neglect of a field of study that is very important in phytopathological research.

The problem has been neglected to an equal degree by the entomologists. Economic entomology, like phytopathology, is relatively young, and workers in this field also, until quite recently, have been engaged in the descriptive aspects of their science. They have been so greatly concerned with the direct injury to plants by insects that they have had very little time for the study of the more obscure injury caused by insects acting as vectors of plant diseases. It is safe to say that the subject of insects in their relation to plant diseases has received no more attention from the economic entomologist than it has from the phytopathologist.

Phytopathology and economic entomology have one large problem in common, namely, the problem of plant protection. Workers in both fields are concerned with protecting our agricultural crops against those innumerable pests which take such a large annual toll from the farm income. In spite of this common interest in a problem of such vital importance to human welfare, the two sciences have widely divergent viewpoints. These different viewpoints can be explained partly by the origin of the two branches of science. Only a few generations ago, biology was considered a highly specialized subject, and it was possible for one man to obtain a fair mastery of the entire field. As science progressed, the sum total of biological knowledge increased so rapidly that one man no longer could master the whole field. Specialization and division of labor became necessary. The first division separated biology into botany and zoology. With the continued rapid growth of science, each of these has since been divided and subdivided into a bewildering number of branches, each branch having its own technique and vocabulary.

Phytopathology is essentially a botanical science whereas economic entomology is a zoological science. Today two young students of biology start out together, but very soon they come to the parting of the ways, one traveling the road of a zoologist, the other that of a botanist. After years of training one becomes a plant pathologist, the other becomes an economic entomologist, and they meet again, faced with the common problem of plant protection. They both realize the necessity of working together and attempt to cooperate, but unfortunately "they don't speak the same language." In the course of their training, their viewpoints have become too widely separated and too greatly specialized.

This situation has resulted in the neglect of a large field of information that is vital to the solution of many problems of plant protection. As the two sciences have advanced steadily, there has been left behind a large "no man's land" in which are many unsolved riddles and unanswered questions. One of the most important of these neglected borderline problems is that of the relation of insects to the spread and development of plant diseases.

Nearly fifty years ago, Waite (1891) first established the fact that insects were of significance as vectors of plant diseases by

demonstrating that fire blight of pears is transmitted by bees. At approximately the same time, it was discovered that insects and other arthropods were of very great importance as vectors of diseases of man and other animals. Yet despite the significance of these early discoveries, progress in the study of insect transmission of plant diseases has been relatively slow. The plant pathologist with his vague knowledge of insects has found it too easy, when faced with an insect-disease complex, to shirk his responsibility by saying, "That's up to the entomologist." In a similar manner, the entomologist has found it equally easy to avoid any problem that might involve a mycological or bacteriological study. It is obvious to one familiar with the phytopathological and entomological literature that this attitude often has delayed for many years the discovery of important facts of insect-plant disease relationships. For the purpose of illustration, it may be well to mention a few examples.

The seed-corn maggot has been studied extensively by entomologists who were well aware of its association with decayed plant tissue, but until recently the insect has been considered as a scavenger attacking only decayed plants (Schoene 1916). In spite of the almost constant association of the insect with potato plants affected with bacterial soft rot, it was completely ignored for many years by all plant pathologists who studied the disease. The failure to recognize the significance of the association can be attributed largely to the prevalence of the idea that the insect lived entirely as a scavenger. It was only when the insect and the bacterial disease were studied together that the true relationship was discovered (Leach 1926).

The bacterial wilt of corn has been studied by plant pathologists since 1897. Early work led to the incorrect conclusion that it was entirely seed-transmitted. Its true methods of transmission were not known until quite recently. Long before it was discovered that the disease is transmitted by the corn flea beetle, the injury caused by this insect was described by entomologists in terms that leave no doubt of the fact that bacterial wilt was present but unrecognized. Forbes (1905), describing the injury to sweet corn caused by the flea beetle in 1891, stated that "whole fields were wilted more or less and some hills entirely killed." This striking injury was attributed to the insect alone without mention of a disease. Metcalf and Flint in 1928

described flea-beetle injury as follows: "The green portion of the leaf is eaten, giving the whole plant a bleached appearance; growth is retarded, and the leaves wilt even during wet weather." Every plant pathologist will recognize this as a good description of the symptoms of bacterial wilt.

The destructive hopperburn of potatoes was confused for many years with tipburn and attributed entirely to the effects of excessive transpiration. In 1918 Ball showed that the greater part of the injury attributed to "tipburn" was caused by the feeding activities of the leaf hopper (*Empoasca fabae* Harris). Had plant pathologists who studied the condition been less reticent in observing the insects, the true cause probably would have been discovered sooner.

The fungi causing blue stain of coniferous wood have been studied by forest pathologists for many years, but it was not until 1929 that these fungi were shown to be pathogenic to living trees. Although their association with the bark beetles had been observed, the significance of the bark beetles as vectors of the fungus was not discovered until an entomologist questioned the ability of the beetles alone to kill the trees so rapidly (Craighead 1928). Work done in response to this question showed that the death of the trees was caused primarily by the blue stain fungus introduced into the sapwood by the bark beetles (Nelson and Beal 1929).

The Need for Cooperative Effort.—The discovery of the nature of virus diseases of plants and the role of insects in the transmission of viruses has greatly stimulated the interest of both entomologist and pathologist in the general subject of insect transmission of plant diseases. The appearance in Europe and America of the destructive and spectacular Dutch elm disease, which is so dependent upon insects for its spread and development, also served to focus the attention of both groups of workers on the problem. The necessity for cooperation between entomologist and plant pathologist in the solution of these borderline problems is now generally recognized by both groups, and the need has been expressed in the literature on numerous occasions. For various reasons, however, the present situation leaves much to be desired. It is one thing to talk of cooperation and another thing to cooperate. In fact, so much has been said and written in recent years about cooperation in research that the word, to

some extent, has fallen into disrepute. Cooperation often works much better on paper than in practice. In order to avoid the difficulties of cooperation, we often plead for coordination of effort. This sounds much better but is often more difficult, for it requires a great deal of earnest cooperation before we can have successful coordination. But despite all these difficulties there is, and has been, a fair amount of real and successful cooperation in scientific research.

The failure or lack of cooperation may be caused by many different factors. The human or personal element is perhaps the most common of them all and the most difficult to overcome. Other causes may be administrative, political or, as indicated above, largely a matter of tradition. However, it is the author's belief that the greatest success in the solution of borderline problems cannot be achieved by the expedient of cooperation alone. Cooperative work is sometimes attempted on the principle of strict division of labor in which all "entomological" work is done by the entomologist and all "phytopathological" work by the phytopathologist. This type of cooperation nearly always is doomed to failure. In the study of the relation of insects to plant diseases, such strict division of labor is not practical. For the greatest success, the invisible, though very real, wall separating the two fields of research must be broken down. This may be rather difficult, but it can be done.

A first step in this direction would be a liberalization of the narrow professional viewpoint, which in effect often hangs out a sign reading, "This is the phytopathological field; entomologists encroach at their own risk" or "This is the entomological field; all phytopathologists keep off." Such a viewpoint may simplify some of the problems of organization, but it is not conducive to the solution of these neglected, but mutually important, problems. The necessity for well-defined fields of research with corresponding responsibility and authority is recognized. Such responsibility and authority are necessary, not only for efficient administration, but also for the existence of the guild spirit so important in scientific research. Nevertheless, when attempts are made to draw too sharp a line between related fields of activity, many problems of vital importance and significance usually are neglected.

A second step would be a modification of our educational procedure so that research workers would be given the viewpoint and training in techniques necessary for the solution of the problem in hand. The worker should have a thorough knowledge of the essentials of both entomological and microbiological techniques. Instead of placing the emphasis upon training entomologists or plant pathologists, some of the workers should be given the training and viewpoint necessary for the solution of this particular kind of problem, namely, the role of insects in the spread and development of plant diseases. It is not proposed that we train mental giants who can master both fields of knowledge, but rather workers who have a sufficient grasp of the essentials of both sciences for the solution of this particular kind of problem. When these qualifications are combined in one man, many of the difficulties of cooperation will be avoided. No claim is made for the novelty of the idea, for already it has met with considerable success in the solution of other borderline problems.

The Complex Nature of the Problem.—The problem of insects in relation to plant diseases is by no means a narrow and restricted field. It includes the study of many questions of broad biologic significance. Insects and plants, including microorganisms, have been closely associated throughout their evolutionary history. In the course of their evolutionary development many different types of insect-plant associations, with varying degrees of complexity, have arisen. Some of these have a direct and fundamental bearing on the problem of disease transmission. For example, the phenomenon of entomophily, or insect pollination of flowering plants, is a biologic association that has much in common with the insect dissemination of plant pathogens. The two associations are strikingly similar in many respects. A student of insect transmission of plant disease will find much of stimulating interest in a study of insect pollination of flowering plants. Similarly, the subject of symbiosis between insects and microorganisms is of vital interest to anyone concerned with the study of insect transmission of plant diseases. There are numerous examples of insect transmission of plant pathogens in which there is a condition of mutualistic symbiosis between the pathogen and its insect vector. For every known association between

insect and pathogenic microorganism, there are numerous similar associations between insects and nonpathogenic microorganisms. The fundamental biological principles are usually the same in both cases. For these reasons, the whole category of interrelationships between plants and insects is considered fundamentally significant to a proper study of the problem of insect transmission of plant diseases. The association of insects with plant pathogens is only one of several such relationships. A perspective of the entire group of insect-plant associations is essential to the best understanding of any particular one.

When insects are concerned in the transmission of a plant disease, three organisms often are involved: the plant (the susceptible), the pathogen, and the insect vector. The association may be, and usually is, more complex than one would conclude from a casual study. For a thorough understanding of the association, the morphology and physiology of all three organisms must be reasonably well understood. Under our present plan of graduate study, very few students of either phytopathology or entomology are sufficiently well grounded in the subjects of plant anatomy, plant physiology, microbiology, insect morphology, and insect physiology to understand clearly the significant biologic processes involved in such an association. Moreover, simple morphological and physiological processes often take on entirely new aspects when involved in such a three-way association.

It is a principle well recognized in plant pathology that, in the study of the epiphytology of a plant disease, one must consider the influence of the environment not only on the susceptible and on the pathogen but also on the interaction of the two. When a third living organism, the insect vector, is introduced, the ecological relations are complicated to a surprising degree, and new kinds of problem arise. The ecological aspect of insect-transmitted plant diseases is a field of investigation that is yet to be exploited extensively. This phase of the insect-transmission problem has been studied more completely in connection with the curly top of sugar beet than any other disease. A study of the ecology of the vegetation on weedy, abandoned farm lands and the problem of overgrazing are subjects that would have had very little interest for plant pathologists in the earlier studies of curly top. Because of their relation to the insect vector of the disease, these studies are now important aspects of the curly-top

investigations. A different type of ecological study is represented also by the work of Fife and Frampton (1936). They showed that the pH gradient from the phloem to the parenchyma of the beet leaf is a factor determining the tissues fed upon by the leaf hoppers. This ecological study has thrown much light on the problem of insect transmission of curly top and may help explain the high degree of specificity often found in insect transmission of viruses.

The study of the more detailed relationships between insect vectors and the diseases transmitted by them includes many problems, the solution of which is definitely limited by the lack of satisfactory techniques. No science can advance more rapidly than the techniques available for its study. The desired technique may be a combination of methods peculiar to microbiology and entomology or something entirely new. These problems of technique offer unique opportunities for those workers who approach them with the proper background of training and experience.

The discovery that insects are concerned in the transmission of plant diseases was made at approximately the same time that it was demonstrated that insects could transmit diseases of man and other animals, but study of the subject has progressed somewhat more rapidly in the field of animal pathology than it has in plant pathology. This difference may be explained in part by the greater value placed upon the life and health of man and his domesticated animals as compared with that of plants. However, the principles involved in insect transmission of the two groups of diseases, in so far as they are comparable, are strikingly similar. The student of insect transmission of plant diseases can learn much from a study of the known facts about insects as they are concerned in the spread of the diseases of man and other animals.

The importance of insects in the transmission of the diseases of man has lead an increasingly large number of investigators into this field of research. These investigators are trained in the fundamentals of both entomology and medical science. They have for their goal the solution of a particular kind of problem, namely, the role of insects in the spread and development of diseases of man. Their work has contributed much to the remarkable advances in medical science in the past few decades. It would

seem that the time is ripe for a similar development in the field of plant pathology.

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CHAPTER II

THE INTERRELATIONSHIPS OF PLANTS AND INSECTS

Plants and insects have inhabited the earth together for millions of years. During this time, each group has undergone extensive evolution. A few relatively simple kinds have given rise to almost innumerable species with a remarkable complexity of form and function. The most important factors in determining the course of this evolution have been the intense struggle for existence and the inevitable natural selection. Plants and insects have lived in close association under conditions that led to many types and degrees of interdependence. Because of the survival value of some of these associations, progressive evolution in the direction of greater interdependence has taken place. Consequently many diverse and complex interrelationships between plants and insects arose, and some of them have survived to the present time.

We are interested here primarily in those associations in which the insect is concerned in some way with the spread or development of plant diseases, but a satisfactory discussion of this relationship depends to some extent upon a knowledge of the other kinds of association. For this reason, a brief analysis of the various interrelationships of plants and insects is given.

The numerous interrelationships of plants and insects may be classified conveniently in the following six groups:

1. Insects that feed upon plants (phytophagous insects).
2. Plants that feed upon insects (entomophagous plants).
3. Plants that cause diseases of insects (entomophthorous plants).
4. Plants that are pollinated by insects (entomophilous plants).
5. Insects and plants that live in symbiosis.
6. Insects that disseminate plant pathogens or aid in the development of plant diseases.

1. PHYTOPHAGOUS INSECTS

This is perhaps the best known and most universal insect-plant relationship. Insects, in common with all other forms of animal

life, are either directly or indirectly dependent upon plants for food. Plants, by virtue of their ability to fix the energy of the sun through the process of photosynthesis, are ultimately the source of the food for all animals. Neither insects nor any other form of animal life could survive if there were no plants. Insects, in feeding upon crop plants, inflict losses amounting to many millions of dollars annually. One needs only to mention such



FIG. 1.—A typical chewing insect, the Mexican bean beetle (*Epilachna corrupta* Mulsant). Five larvae and one adult beetle feeding on a bean leaf.

examples as the grasshopper, the chinch bug, cutworms, and white grubs to picture this aspect of the relationship. Insects feed not only on living plants but also on all types of stored plant product. All kinds of plant from the smallest microorganism to the largest tree are utilized by them. Insects feed in many different ways. Some, such as grasshoppers and beetles, chew the plant tissues and digest them (Fig. 1); others, such as aphids, leaf hoppers, and chinch bugs, puncture the plants and suck the sap from the living cells (Fig. 2); still others, including bees, wasps, and butterflies, feed upon nectar and other plant exudates (Fig. 3). Whatever the method of feeding, the insects are usually

highly specialized for the utilization of some particular kind of plant or plant product.

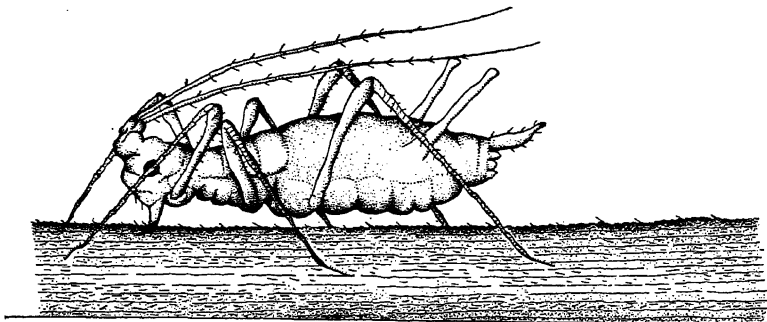


FIG. 2.—A sucking insect, an aphid, feeding on the mid-vein of a potato leaf. The insect's beak is penetrating deep into the tissues of the plant from which cell sap is being drawn. (After Houser, Guyton, and Lowry.)

In addition to the direct destruction of tissue by feeding, many phytophagous insects indirectly destroy plants by breeding



FIG. 3.—A honeybee sipping nectar from an apple blossom. A representative of a large group of insects that feed upon plant products without wounding the plant tissues.

in the tissues. Some species spend much of their life span boring or mining in some part of the plant and often cause as much

damage in connection with breeding operations as they do by direct feeding (Fig. 4).

Some insects are *polyphagous*, feeding upon an almost unlimited number of plant species. Others are *oligophagous*, feeding upon a limited number of closely related plant species. Still others, such as certain species of aphids, are *polyphagous* during one season or one stage of development and *oligophagous* during the others. A few insects restrict their feeding to a single species or variety of plant and are classed as *monophagous*. The number



FIG. 4.—The brood gallery of the pine bark beetle (*Ips pini*) in the sapwood of a red pine tree.

of different insect species that may feed upon a single plant species varies widely. Some plants such as poison ivy [*Rhus toricodendron* (Tourn.) Mill.] serve as food for less than half a dozen insect species, whereas others, for example, the oak and apple, are hosts for four or five hundred different species.

The injury caused by insects that attack cultivated crop plants is so great that one often overlooks the fact that some insects feed upon and destroy noxious plants. Those feeding upon the various species of cactus (*Opuntia* spp.) have rendered notable service in the control of these plants in Australia (Prickly-Pear Land Commission, 1925 to 1932). Several species of *Opuntia* were introduced into Australia about 1840 as ornamental plants.

They escaped and multiplied as weeds so rapidly that by 1870 they were out of control. Largely because of the absence of natural enemies, they continued to spread until more than 60 million acres of land were made unfit for agricultural purposes. The cactus plants, especially those of *Opuntia inermis* and *O. stricta*, grew in such dense masses that all ordinary cultural practices were ineffective in preventing their spread. The insect enemies of *Opuntia* were then introduced from Texas and Argen-



FIG. 5.—A dense infestation of prickly pear (*Opuntia inermis*) before subjected to the attack of the insect, *Cactoblastis cactorum*. Compare with Fig. 6. (After Dodd.)

tina, and they, together with the microorganisms that they transmit, have been so effective that approximately twenty-five million acres have been reclaimed, and further spread of the cactus has been checked (Dodd 1927, 1933, 1936). Of the insects introduced, the Argentine moth borer (*Cactoblastis cactorum* Berg.) has been by far the most effective (Figs. 5 and 6).

This outstanding success of cactus control by insects in Australia has focused the attention of entomologists upon the

influence of insects on the growth of weeds under natural conditions and the possible use of insects in the control of other noxious weeds. In general, the most noxious weeds are those which have been imported into a new region where their insect enemies have not become established. Often they are not considered noxious in their native habitat, and it is entirely possible that



FIG. 6.—The same area of prickly-pear infestation shown in Fig. 5 three years later, after infestation with *Cactoblastis cactorum*. The prickly pear plants have been destroyed by the insect and the microorganisms transmitted by it; insert, adult and larva of *C. cactorum*. (After Dodd.)

their noxious character would be greatly decreased if the insects and other pests that feed upon them in their native home were introduced to the new locality. This possibility has been investigated only superficially but is worthy of further study.

Insects are also important scavengers and consume many kinds of dead plant tissue that would otherwise accumulate in excessive amounts. Ants are said to be of especial importance in this

respect in the tropics. Insects and fungi usually are closely associated in the destruction and decomposition of plant remains.

2. ENTOMOPHAGOUS PLANTS

A number of plant species reverse the usual order of things by feeding upon insects. Plants that feed upon insects are by no means so numerous as insects that consume plants, yet at least five hundred species of entomophagous plants are known (Kerner and Oliver 1895). Charles Darwin was among the first to study these plants, having published an extensive account of them in 1875. This unusual plant-insect relationship is primarily of academic interest, for the number and kind of insect consumed by plants are of little economic significance to man. It is of interest chiefly for the intricate and highly specialized anatomical and physiological adaptations possessed by the plants, which enable them to trap the insects and to digest them. It illustrates the extent of interdependence between insects and plants and indicates the range of possibility in the evolutionary development of the interrelations between insects and plant pathogens.

The mechanisms used by plants in trapping insects are of several different types. For convenience, these may be classified into three general groups. One group captures insects by means of sticky exudates on the leaf surfaces. A second group entraps the insects mechanically by various structural modifications and involves no unusual movement of the plant parts. The third group entraps the insects by special movements of modified leaves.

The **flycatcher** (*Drosophyllum spp.*) is a good example of the first group. The stems of this plant are covered with stalked glands that secrete dewlike drops of a very sticky fluid. Insects attracted by this exudate alight upon the stems, become entangled in the sticky fluid, and cannot escape (Fig. 7). After an insect is caught, a second type of gland secretes an acid fluid, rich in enzymes, which dissolves and digests the entire insect body with the exception of the chitinous exoskeleton. The secreted digestive fluids, along with the dissolved insect body, are then reabsorbed by the plant cells. The sticky fluid also is temporarily reabsorbed, and the chitinous remains of the insect fall off or are blown away by the wind. This plant grows in dry places in Portugal and Morocco, where the natives have long

used it in catching flies, its action being based on the principle of the well-known "tanglefoot."

The pitcher plants (*Sarracenia*, *Darlingtonia*, *Nepenthes*, etc.) entrap insects by special anatomical adaptations, working on the principle of a pitfall. The pitfall is formed by elaborate modifi-

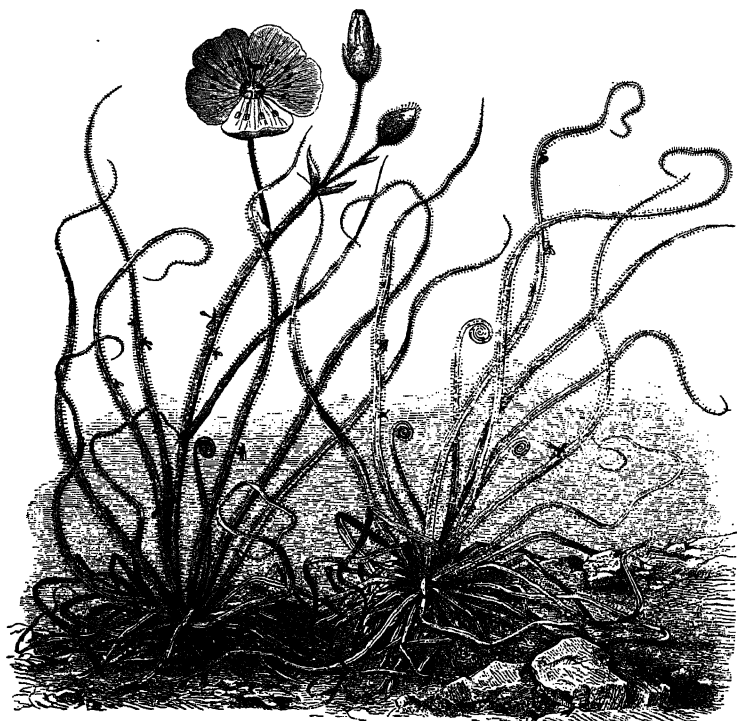


FIG. 7.—The flycatcher (*Drosophyllum lusitanicum*) with insects entangled in the sticky exudate. (After Kerner and Oliver.)

cations of the leaf petioles and assumes a great variety of forms (Fig. 8). The leaf lamina is usually small and often forms a canopylike covering over the mouth of the pitfall. The pitfall consists of three essential parts: (1) a device for allurement of insects, (2) an arrangement for entrapping the insects and for preventing their escape, and (3) a means of digesting the prey.

The allurement is accomplished by means similar to those used by entomophilous plants to attract insects to flowers. Near the opening of the "pitcher," or pitfall, there is often a spot of bright color, usually purple, yellow, green, or white. Drops of nectar are secreted around the rim of the pitcher, and there is often a nectar-baited pathway, from the ground to the mouth of the

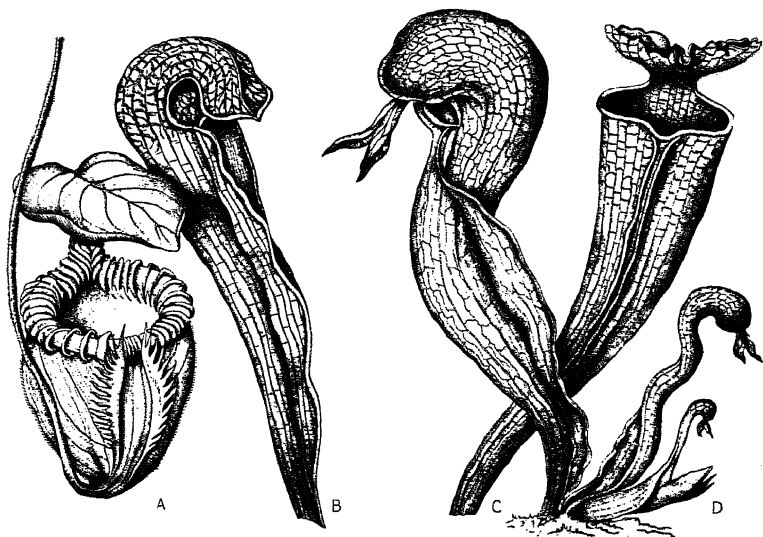


FIG. 8.—Pitcher plants. A, *Nepenthes villosa*; B, *Sarracenia variolaris*; C, *Darlingtonia californica*; D, *S. lacinata*. The petioles are modified to function as pitfalls in which insects are entrapped. They are usually partly filled with a liquid rich in proteolytic enzymes that digest the insects. (After Kerner and Oliver.)

pitcher, that attracts wingless insects and leads them to the pitfall.

The entrapping device consists of a variety of special structures. The inside of the rim of the pitcher is usually formed by a series of smooth bristles pointing downward. The inside walls are often lined with similar bristles or with sticky glandular hairs (Fig. 9). There is always a quantity of liquid in the bottom of the pitcher into which the insects fall and eventually drown. The modified canopylike leaf lamina cuts off the direct light from the opening, adding to the difficulty of escape.

The means of digestion have not been determined for all species of pitcher plants. In some species, the presence of pepsin and acid has been demonstrated. Digestive enzymes are probably secreted by most species; but putrefactive bacteria are often found in the liquid, and it is probable that they also are involved in the digestion of trapped insects.

The butterworts (*Pinguicula spp.*) catch insects in a manner similar to that of *Drosophyllum* except that a definite movement of the leaves is involved. There are about forty species of this plant, found mostly in moist locations in arctic and subarctic regions. The plant grows as a rosette with flat oblong leaves

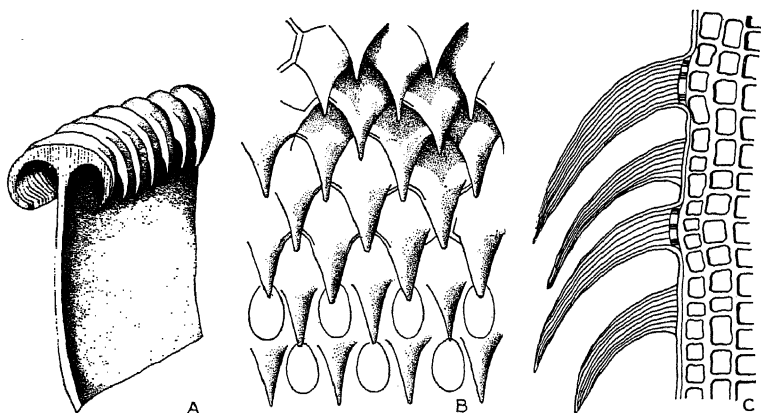


FIG. 9.—Downward-pointing spines found along the inner walls of the pitfalls of various species of pitcher plant. (After Kerner and Oliver.)

lying prostrate on the ground. The upper surface of each leaf is covered with two types of gland, a stalked one secreting a sticky fluid that attracts and holds the insect and a sessile one that secretes the digestive fluid (Fig. 10). The latter fluid is secreted as a direct response to the specific stimulus of a foreign nitrogenous substance present on the leaf. After the insect is caught, the edges of the leaves usually curl upward and inward, covering the smaller insects or pushing the larger ones toward the center of the leaf where the glands are more numerous. The digestive fluid is rich in the enzyme causing rennet coagulation of milk. The plant is said to have been used by the Laplanders as an addition to milk in making a popular milk delicacy.

The sundews, comprising the various species of *Drosera*, have a very delicate mechanism for entrapping insects. The insects

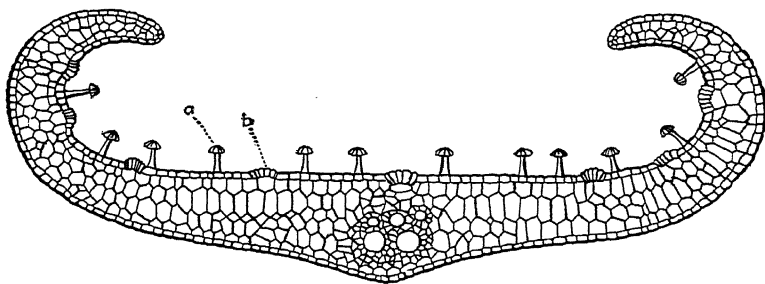


FIG. 10.—A diagrammatic cross section of a butterwort leaf showing the stalked glands (a), which secrete the sticky fluid that entangles the insect. When an insect is entrapped, the margins of the leaf curl inward toward the mid-vein, and digestive enzymes are secreted by a second set of glands (b). (After Kerner and Oliver.)

are trapped by leaves that grow as a rosette and remain prostrate on the ground (Fig. 11). Each leaf of the rosette is broad and spatulate at the tip. The upper surface of the broad part of the leaf is covered with about two hundred red tentaclelike filaments, each with a drop of glistening sticky liquid at the apex (Fig. 12a). Insects attracted to the glistening drops alight on the leaf and become entangled in the fluid. As soon as the insect is caught, the tentacles start bending inward in a way that brings their tips with the sticky liquid in contact with the prey. Within a few minutes, all the tentacles are pointing inward and touching the insect (Fig. 12b, c, and d). The sticky fluid is secreted in increasing amounts, and the insect is soon completely covered. This movement takes place only in

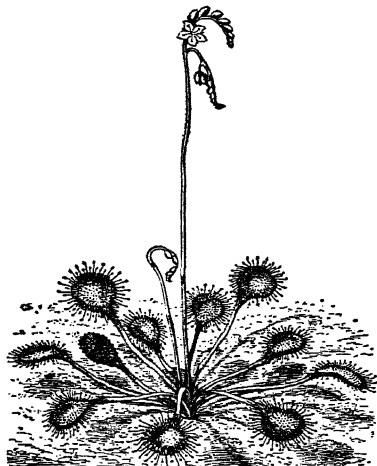


FIG. 11.—A plant of the Sundew (*Drosera* sp.) showing the rosette of spatulate leaves that catch and digest insects. (After Brown, by courtesy of Ginn and Company.)

response to a nitrogenous substance. The response is extremely specific, as the tentacles will not respond to such objects as sand, raindrops, or other nonnitrogenous matter, yet $\frac{1}{4000}$ part of a

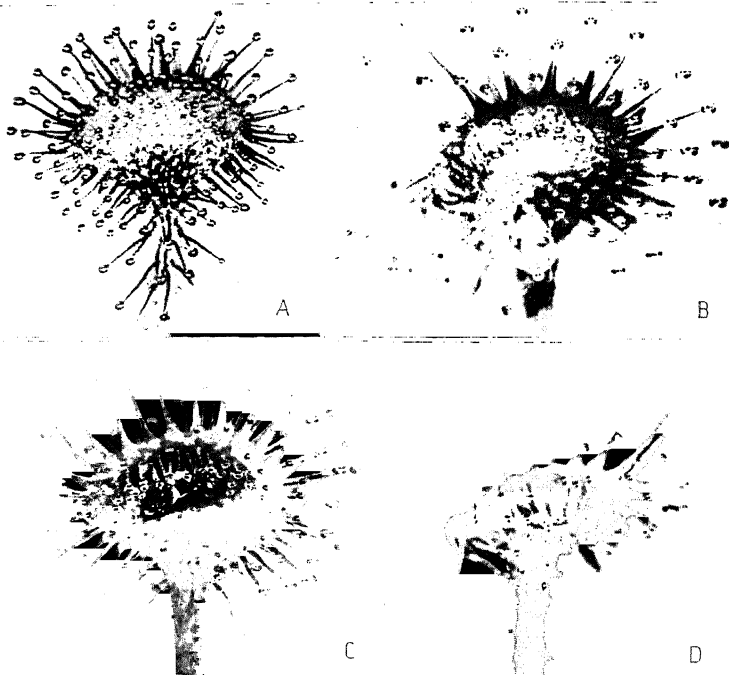


FIG. 12.—Leaves of the sundew (*Drosera*) showing the method of entrapping insects. A, a leaf with the tentacles fully expanded, each tentacle bearing a drop of sticky exudate at the tip; B and C, leaves showing stages in the capture of an insect. In B, the insect has alighted on leaf and is becoming entangled in the sticky exudate. Note the tentacles bending inward moving the insect toward the center. In C, the insect has become thoroughly covered moving the insect toward the center and has ceased to struggle. In the final stages, D, the tentacles all bend inward completely enclosing the insect which is digested by enzymes in the secreted liquid. Approx. 3 X. (From the instructional sound film, "Plant Traps," produced by Erpi Classroom Films, Inc.)

milligram of ammonium carbonate will cause them to bend. When the insect victims have been digested, the tentacles straighten out and resume their original position.

The bladderworts (*Utricularia* spp.) catch insects and other small water animals in a valvular trap. They are mostly rootless

water plants that float near the surface of the water in summer and sink to the bottom as dormant buds in winter. Along both sides of the long lateral stems are borne bladderlike structures open at one end (Fig. 13). The open end is surrounded by bristles in position to form a funnel directing the victims toward

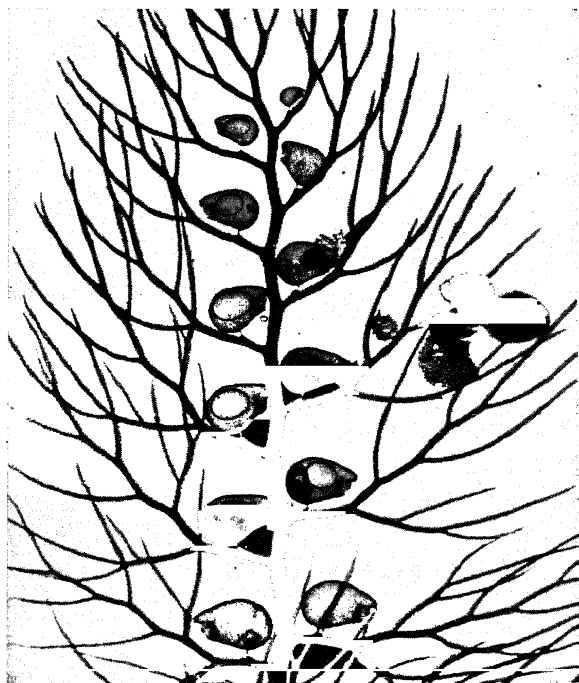


FIG. 13.—A portion of a branch of bladderwort (*Utricularia vulgaris*). Five of 10 active bladders contain mosquito larvae. (After Matheson.)

the entrance. The opening is fitted with a valvelike trap door through which the insects or other organisms are drawn. The mechanism of the trap of *Utricularia* has been studied extensively by several workers, but the investigations of Lloyd (1929, 1932, 1933, and 1935) are the most exhaustive and conclusive. Earlier writers interpreted the trapping mechanism as a simple valvular trap door through which the insects forced their way and then

were unable to escape (Kerner and Oliver 1895), but later work has shown the mechanism to be of a more complex nature.

According to Lloyd, the trap door, when in a set position, rests upon the outer edge of a threshold and forms a watertight seal (Fig. 14). The semipermeable walls of the bladder pump the water out of the lumen, a negative pressure within being thus formed. This negative pressure is sufficient to cause the walls to bend inward, and the pressure also holds the door firmly in

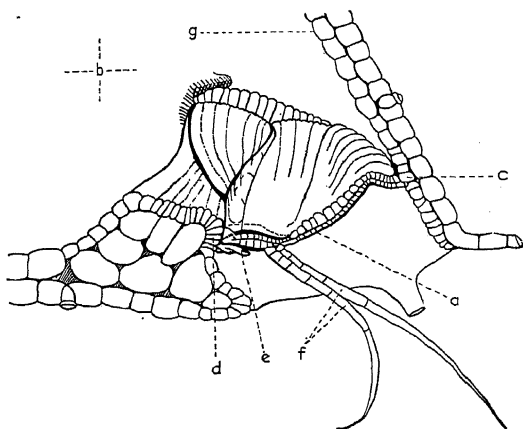


FIG. 14.—Diagrammatic sketch of the bladder of *Utricularia* showing the mechanism of the trap. *a*, door of bladder; *b*, interior of bladder; *c*, attachment of door to wall of bladder; *d*, threshold; *e*, exfoliated cuticle that makes a watertight seal between door and threshold; *f*, trigger hairs. When the insect pushes down on the trigger hairs, they act as a lever, raising the free edge of the door above the threshold, and break the seal. The pressure of the water forces the door inward and upward against the roof (*g*), and the insect is sucked in with a stream of water. (After Lloyd.)

place. The seal of the door is made watertight by a thin veil of exfoliated cuticle on the threshold against which the door rests. Three or four stiff bristles arise from a thickened plate near the middle part of the lower free edge of the door and point outward. These bristles are attached at such an angle that a slight movement of their free ends exerts sufficient leverage to raise the lower edge of the door and break the seal. When this occurs, the pressure of the water from without forces the door inward, and the walls of the bladder expand to their normal shape. A stream of water is drawn into the bladder, and the insect that

moved the trigger hairs is drawn in with it. As soon as the bladder is filled with water, the door slowly returns to its original position, and the walls start pumping the water out until the set condition is again reached. This usually requires about 15 to 30 minutes. The trigger hairs, according to Lloyd (1932), are entirely mechanical in their action, operating as levers to lift the bottom edge of the door and break the watertight seal. The response is not one of irritation as in the Venus's-flytrap.

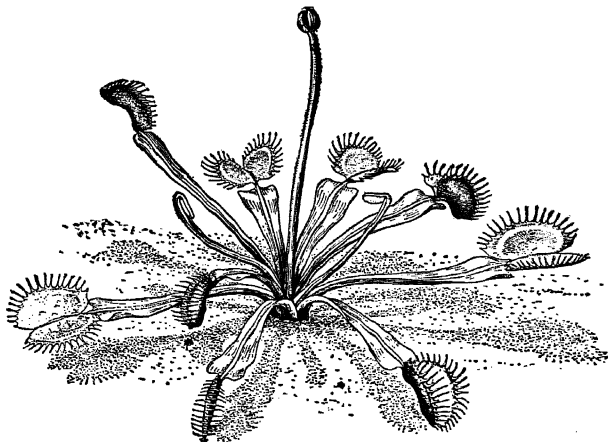


FIG. 15.—Venus's-flytrap (*Dionaea muscipula*). Approx. $\frac{1}{3}$ X. (After Brown, by courtesy of Ginn and Company.)

The inside walls of the trap are supplied with glands that secrete the usual proteolytic fluid to digest the insects. In addition to insects, many other aquatic animals are caught and consumed. Mosquito larvae are among the most common insects caught by the bladderworts, and it is claimed by some observers (Matheson 1930) that these plants play an important part in mosquito control in certain localities, thus aiding in the control of malaria.

The Venus's-flytrap (*Dionaea muscipula*) is perhaps the most striking of all the entomophagous plants. This plant occurs in nature only in a narrow strip of country along the east coast of North America in and near peat bogs. It, like the sundew, grows in the form of a rosette (Fig. 15). Each leaf consists of a flat spatulate petiole and a roundish lamina divided by the mid-rib

into two symmetrical halves inclined to each other at an angle of 60 to 90 degrees, like a half-open book. The margins of the lamina bear from ten to twenty long sharp spines. In the center of the upper surface of each half of the leaf, there are three shorter spines, or trigger hairs. The entire upper surfaces are covered with still shorter glandular structures that are colored purple and are capable of secreting a sticky liquid (Fig. 16).

When the insect, attracted to the leaf by the purple color and the sticky liquid, touches one of the six spines in the center of the leaves, the two lamina fold together tightly and the insect is entrapped (Fig. 16*B*). The movement is rather slow, 10 to 30

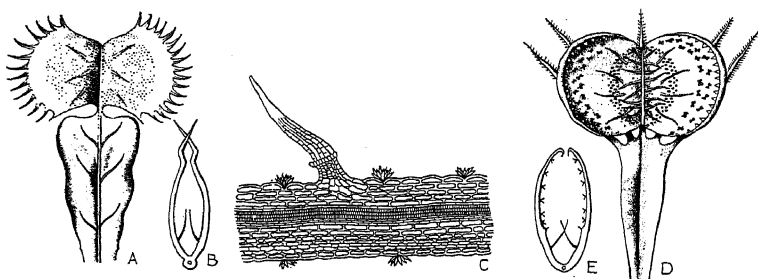


FIG. 16.—The entrapping apparatus of Venus's-flytrap and the closely related *Aldrovandia*. A, an expanded leaf of Venus's-flytrap; B, a cross section of a closed leaf; C, one of the sensitive bristles of the upper surface of the leaf; D, an expanded leaf of *Aldrovandia*; E, section of a closed leaf. (After Kerner and Oliver.)

seconds being required for the trap to close completely; but the interlocking spines along the margins prevent the escape of the insect, and the trap is very effective. As soon as the insect is trapped, the glands begin secreting the digestive fluids. The leaf remains closed until the soft tissues of the insect body are completely digested and the liquid reabsorbed, a process that may require two weeks or more. The leaf then opens and is soon ready to entrap another insect.

These examples of entomophagous plants will suffice to illustrate the exceedingly complex and intricate nature of this type of interrelationship between plants and insects. It is evident that the entomophagous habit of these plants must have arisen through a long process of evolution. It is significant that practically all species of entomophagous plant are found in nature on soils deficient in available nitrogen. In all probability, the

need of a richer source of nitrogen by the plants has been the directing influence in the origin of the relationship.

Nematode-entrapping Fungi.—Although nematodes are not insects, they may serve as vectors of pathogenic fungi as pointed out in Chap. XI. In view of this fact, it is of interest to know that certain fungi have evolved special adaptations for entrapping and consuming nematodes. Sherbakoff (1933) has described a remarkable soil fungus (*Anulospodium nematogenum*) with ring-shaped spores that function as nematode traps. The ring-shaped conidia that are set free in the soil have a diameter somewhat less than that of a nematode's body. The nematode in moving through the soil gets a ring over the tapering anterior part of its body but is unable to pass entirely through it. By the movement of the nematode the ring is pushed farther and farther back over its tapering body, forming a distinct constriction. The conidia then germinate and send hyphae into the nematode's body which is rapidly consumed by the fungus. Further studies of related nematode-entrapping fungi have been made by Drechsler (1933*a*, *b*, *c*, 1934, 1935, 1936), Couch (1937), Linford and Oliveira (1938), and Linford and Yap (1939).

3. ENTOMOPHTHOUS PLANTS

Insects, like other forms of life, are subject to disease. In general, the diseases of insects are caused by the same kinds of microorganism that affect other animals or plants, namely, bacteria, fungi, viruses, and protozoa. The bacteria and fungi are plants, the nature of the viruses is not definitely known, and the protozoa belong to the animal kingdom. Some of the plants that cause diseases of insects affect injurious species and benefit man by aiding him in the control of these pests. Other diseases attack such beneficial insects as the honeybee or the silkworm and are detrimental to the interests of man. The diseases of insects are so numerous that a discussion of them all is not practical here. But with the view of illustrating another insect and plant relationship, a few representatives will be described. For more extensive treatments of the subject, see Paillot (1933) and Sweetman (1936).

Bacterial Diseases of Insects.—The best known bacterial diseases of insects are probably those that affect the honeybee. European foul brood is an infectious septicemic disease of honey-

bee larvae and is caused by *Bacillus alvei* Cheshire and Cheyne. Affected larvae become soft and lose their normal shape and color. At first they turn yellow or gray and eventually dark brown. The internal tissues are transformed into a viscid liquid. American foul brood of bees is a similar disease caused by *Bacillus larvae* White. Both diseases are of considerable economic importance causing much loss to the beekeeper of both Europe and America (White 1920a, b). Diseases of similar nature have been described in a few instances as affecting injurious insects. *Bacillus sphingidis* White is credited by White (1924a) with causing a disease of this nature on the tobacco hornworm (*Protoparce sexta* Johan.) and the tomato hornworm (*P. quinquemaculata* Haw.). A septicemia of cutworms caused by *Proteus noctuorum* (White) Bergey *et al.* has been described also by White (1924b).

A disease of this general type was discovered on grasshoppers by D'Herelle (1911, 1912) who described the bacterial pathogen and named it *Coccobacillus acridiorum*. D'Herelle advocated the use of the disease in controlling grasshoppers. It was claimed that the cultures could be made more virulent by successive passage through grasshoppers inoculated by needle injection. Suspensions of the virulent bacteria were used in spraying infested fields and the advance ranks of the migratory hoppers. Quick and successful results were claimed by D'Herelle, but extensive trials made later in many parts of the world have failed to demonstrate the effectiveness of this method of grasshopper control (Kraus 1916). Barber and Jones (1915), who were unable to verify the claims of D'Herelle, demonstrated that the bacteria were virulent when injected into grasshoppers but that they were not effective when sprayed upon the insects without wounding.

Fungus Diseases of Insects.—A relatively large number of fungi are known to cause diseases of insects. Only a very few of them can be discussed here. Fungi pathogenic to insects are found in the Phycmycetes, Ascomycetes, and Fungi Imperfecti. The Basidiomycetes apparently contain no insect pathogens of importance. The Phycmycetes include one order, the Entomophthorales, all members of which are exclusively parasitic on insects. One of the best-known genera of this order of entomophthorous fungi is *Empusa* which contains about forty species, mostly parasitic on flies. *Empusa muscae* Cohn is the species

affecting the common housefly. The disease appears most frequently in late summer and fall when the weather is cool and the vitality of the fly is low. At this time the parasitized and dead flies may be observed adhering to window panes and other objects surrounded by a halo of white translucent spores that have been shot off from the fungus growing in the body of the insect (Fig. 17). The spores are sticky and adhere to any object they touch, including the bodies of other insects. The spores germinate, and the mycelium penetrates directly through the cuticle of the insect and reaches the blood cavity where hyphal segments are formed. The segments are dispersed in the blood throughout the body until the entire internal parts are destroyed.



FIG. 17.—A fly killed by *Empusa muscae*. The dead fly, stuck to a pane of glass by the fungus, is surrounded by a white zone of spores that have been shot off. (From Sweetman, "Biological Control of Insects," by permission of the Comstock Publishing Company.)

The spores are formed on the surface of the insect and forcibly abjected from the sporophores.

Grasshoppers, aphids, and other insects are often parasitized by species of *Entomophthora*. These fungi usually attack the insects when the weather is both hot and humid and when the insects are overcrowded. Under such favorable conditions the disease may spread rapidly, killing large numbers of insects, and it is probably an important factor in natural control of many insect species. Periodic attempts have been made to use the fungus in artificial control of grasshoppers. The fungus has been cultured and sold in tubes for use by spraying on insects in the field. The attempts have been based on the theory that if an epizootic is started, the disease will effectively destroy the insects in sufficient numbers to effect practical control. In practice, however, the attempts have met with very little success.

There is abundant evidence that naturally occurring epizootics of insect diseases serve as important means of natural control, but man has not been very successful in producing artificial epizootics. Claims of success usually have been based on insufficient evidence, and in most cases the method has not withstood careful and critical investigation. In general, it has been found that the fungus is normally present in sufficient quantity to start an outbreak if other conditions (high temperature, high humidity, and overcrowding) are favorable. If these conditions are not favorable, the application of additional inoculum is of little or no avail. Numerous attempts have been made with many different diseases of insects, but this has been the conclusion reached in nearly all careful efforts to evaluate the effectiveness of this method of insect control. The most promising outlook for the use of insect diseases in the control of insects is by introducing the insect pathogens into a locality where they do not normally occur.

In certain sections of Florida, several species of fungus are very important in the natural control of the citrus white flies [*Dialeurodes citri* (Riley and How.) and *D. citrifolii* (Morg.)]. The abundant rainfall and high temperatures in summer are very favorable for fungus development, and the summer broods of the insects usually are kept under control (Morrill and Back 1912). The importance of the fungi in the control of the insects is evident when the citrus trees are sprayed with Bordeaux mixture. The spray controls the fungi, and the white flies increase with abnormal rapidity. The two most important fungi are *Aschersonia aleyrodidis* Webber and *A. goldiana* Sacc. and Ellis. The former, known as the "red" *Aschersonia*, attacks both species of white fly, while the latter, known as the "yellow" *Aschersonia*, affects only *D. citrifolii*. In 1915, the Florida Plant Board began furnishing cultures of these fungi to the growers for use in infesting the orchards to increase the efficiency of control. Infestations may be started in an orchard either by spraying the trees with a suspension of the fungus spores or by pinning leaves with infested insects to the leaves of noninfested trees (Watson and Berger 1932).

One of the more recently reported efforts to control an insect pest by a fungus disease is that of Dustan (1924), dealing with the control of the European apple sucker *Psylla mali* by *Ento-*

mophthora sphaerosperma. The European apple sucker is not a native of North America and was first observed in 1919 in Nova Scotia, where it proved to be destructive in apple orchards. In the following year, local epizootics of the fungus diseases were observed. In orchards where the disease was present, the insects were so reduced in numbers that little injury was caused to the apple trees. In 1922 and in 1923, attempts to hasten the spread of the disease were made by introducing inoculum in the form of diseased insects into orchards where the disease had not been observed. It was stated:

Usually about a week after a "planting" had been made the fungus began to appear in the orchards under experiment. Once started it spread very quickly and within two or three weeks, depending, of course, on the weather conditions, was generally distributed throughout the orchards. At this time no disease had appeared in the checks, proving conclusively that the outbreaks in question were due solely to the artificial spread of the fungus.

The success in this case led to the recommendation that such "plantings" of diseased material be made by those orchardists who were troubled by the apple sucker. The success obtained by Dustan may possibly be explained by the fact that the insect was a recent importation into North America and that on account of unfavorable weather conditions the spread of the disease had not kept pace with the spread of the insect. If this were true, his "plantings" would have introduced the disease into regions where it had not previously been established. In any event, it appears that the disease is an important factor in the natural control of the insect, and if it were not present, the insect would cause much greater damage than it does.

Among the Ascomycetes, the genus *Cordyceps* contains about two hundred species, most of which are parasitic upon insects. The fungus mycelium permeates the entire body of the insect and forms aerial hyphae and conidia over the surface. The conidia vary with the species and may be of the *Penicillium*, *Verticillium*, or *Isaria* type. The diseases caused by these fungi are often referred to as the "Muscardin" diseases.

The mycelium within the body of the insect is eventually transformed into a sclerotium that keeps the approximate form of the insect. The fungus survives the winter in this stage usually in, or on the surface of the soil, and in the spring a stalk-

like stroma arises from the sclerotium (Fig. 18). The perithecia are borne embedded in enlargements at the end of the stroma, from which the ascospores are liberated into the air. A species of *Cordyceps* is often found on the white grubs (*Phyllophagus spp.*) and may be a natural control factor in dense populations.

The Laboulbeniales of the Ascomycetes include a group of highly specialized fungi, all obligate parasites on insects. They are of academic interest only. They are not fatal to the insect

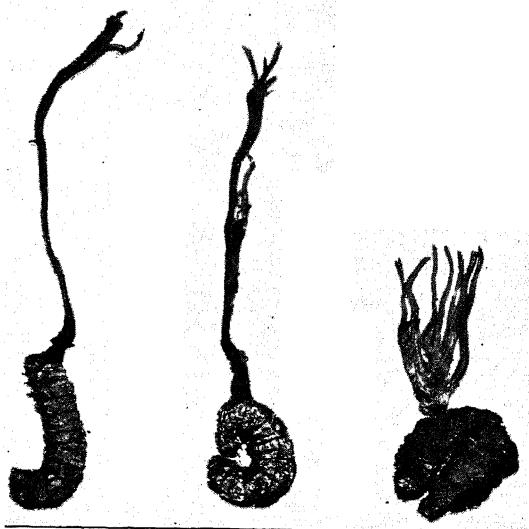


FIG. 18.—*Cordyceps sp.* on larvae of the codling moth showing the stalklike stromata growing from the dead insect. (After Metcalf and Flint.)

and apparently cause little or no injury. The fungi do not penetrate the insect body but are fastened to the integument by small "holdfasts" and derive their nourishment through minute holes in the body integument. They form small hairy or velvety patches on various parts of the insect's body. They are often highly specialized as to the particular part of the body affected, some species being confined entirely to one segment or to certain legs of a single species of insect.

The vegetative mycelium is much reduced, and the reproduction is entirely by ascospores, a highly specialized type of sexual

reproduction similar in many ways to that of the red seaweeds (Florideae). Some species are homothallic, and others are heterothallic.

Infection occurs only when one insect comes in close contact with an infected one, most commonly during copulation. The ascospores, covered with a sticky matrix, are forced out of the perithecia and adhere to the insect's body. A holdfast is formed, and when food relations are established the spore divides and grows into its mature differentiated structure. The best and most extensive account of these interesting fungi is that of Thaxter (1896, 1908).

Virus Diseases of Insects.—Some of the most virulent diseases of insects are caused by viruses, Lepidopterous species being especially subject to them. The best known virus diseases of insects are those affecting the silkworm (*Bombyx mori* L.), the gypsy moth (*Porthetria dispar* L.), and the nun moth (*Lymantria monacha* L.). The wilt disease (*grasserie* or *Gilbsucht*) of silkworms, is extremely destructive and is often a limiting factor in silkworm culture. The similar wilt diseases of the nun moth and of the gypsy moth also occur in epizootic proportions and constitute important factors in the natural control of these insect pests.

Caterpillars affected with wilt become flaccid, and the internal organs are completely liquefied. The affected tissues are characterized by the presence of numerous very small refractive, crystal-like polyhedral bodies from which the term "polyhedral diseases" has arisen. These bodies are protein in nature and may be compound crystals of the infective agent. They have been concentrated by centrifuging and washing but have retained their infectious nature. The infectious agent, in common with other viruses, can be passed readily through certain bacteriological filters.

The caterpillars may become infected by ingesting virus-contaminated material or by direct subcutaneous inoculation. In nature, the former is the more frequent method of infection, although there is some evidence that the diseases are transmitted by parasitic or predacious insects and mites (Allen 1916). There is some evidence for the conclusion that the virus of *grasserie* may be transmitted through the eggs of the silkworm, although this theory is not accepted by all investigators.

Efforts to control noxious insects by the production of epizootics through artificial inoculation with viruses have met with the same doubtful success that has characterized similar efforts with the bacterial and fungus diseases. However, the extent to which other control measures are necessary may be determined by the prevalence of the disease.

Protozoal Diseases of Insects.—Although bacteria, fungi, and viruses are the principal causes of insect diseases, there are several well-defined diseases of insects caused by protozoa, the best known of which is probably that affecting the silkworm, caused by *Nosema bombycis* Nag. and commonly known as "pebrine." The mode of parasitism of the protozoa and the methods by which they are transmitted vary extensively with the different diseases. There is evidence of transmission of some of the protozoal diseases by parasitic or predacious insects. Payne (1933) has reported the transmission of a disease of the Mediterranean flour moth (*Ephestia kühniella* Zeller) by a Hymenopterous parasite (*Microbracon heinator* Say). The disease is not transmitted by ingestion and is not infectious in the absence of the parasite. Furthermore, the first point of infection is in the ganglion which the parasitic wasp pierces in the act of oviposition. By ovipositing in one larva after another, the wasp becomes especially effective as a vector of the disease.

4. ENTOMOPHILOUS PLANTS

One of the most highly developed and perhaps the most interesting association of plants and insects is that of entomophily, or insect pollination of flowering plants. The association has been of great significance in the evolution of plant species and has contributed to the development of many highly specialized relationships between plants and insects. As will be pointed out in some detail in Chap. IV, there are many points of similarity between insect pollination and insect transmission of plant diseases, and a knowledge of the relationships involved in entomophily is helpful in a study of the problem of insect transmission of plant diseases.

Pollination is the act of transferring the pollen from the anther of a flower to the stigma of the same or another flower and is necessary for fertilization and seed production in practically all seed plants, although there are a few species in which

parthenogenesis occurs. The transfer of pollen from the anthers of a given flower to the stigma of the same flower is termed *self-pollination*. When the transfer is from one flower to another on the same plant, it is called *close pollination*, while *cross-pollination* is the transfer of pollen from the flowers of one plant to the stigmas of flowers on another plant. Plants whose flowers can be fertilized by self-pollination or close pollination are said to be *self-fertile*. Some plants are *self-sterile*, a condition in which the flowers can be fertilized only by cross-pollination. It is obvious that when cross-pollination is the rule the probability of hybridization is increased.

Some plants have imperfect flowers, one kind of flower (staminate) producing fertile pollen but having no normal ovaries, another kind (pistillate) having normal ovaries but no pollen. These two kinds of imperfect flower may occur on the same plant (monoecious) or on different plants (dioecious).

Cross-pollination stimulates variation by increasing the frequency of hybridization. As variation is a primary factor in evolution, cross-pollination has been a potent factor in the evolution of the flowering plants. Insects, as one of the principal agents of pollination, have played an important part in this evolutionary process, and many of the adaptations of flowers to insect pollination are concerned with means of ensuring cross-pollination. In other words, the phenomenon of entomophily has had a great influence on the evolution of flowering plants because, by favoring cross-pollination, it has increased the frequency of hybridization and variation. The increased frequency of variation acted upon by natural selection has resulted in the evolution of new adaptations for insect pollination, and these, in turn, have further facilitated cross-pollination. Thus, there is a never-ending cycle that has been a very potent factor in evolutionary development of both plant and insect life.

Pollination is accomplished in several different ways and plants are sometimes classified on the basis of the principal agent of pollination as follows:

1. Hydrophilous—water-pollinated.
2. Anemophilous—wind-pollinated.
3. Zodiophilous—animal-pollinated.
 - a. Entomophilous—insect-pollinated.
 - b. Malocophilous—snail- or slug-pollinated.

- c. Ornithophilous—bird-pollinated.
- d. Chiropterophilous—bat-pollinated.

The anemophilous and entomophilous groups include by far the largest number of species.

The Origin of Entomophily.—Although there is evidence that some of the primitive angiosperms were insect-pollinated and that certain modern anemophilous species may have arisen from entomophilous forms (Hutchinson 1926), it is generally agreed that the most primitive flowering land plants were wind-pollinated. When pollen grains are disseminated by the wind, pollination is accomplished only when, by chance, a pollen grain falls upon a stigma of a plant of the proper species. Considering the small size of the stigmatic surfaces and the separation of the pollen grains as the distance from the source of supply increases, it is obvious that there is a tremendous waste of pollen. In general, the wind-pollinated species produce very large quantities of pollen. This is true of present-day species, and the abundance of fossilized pollen in the geological records of the Carboniferous age indicates that this was true also of the primitive wind-pollinated plants of that period.

Early in the evolutionary development of plants, insects discovered the food value of pollen and began regular visits to the source of supply. Some of the pollen stuck to the bodies of the insects and was transported to other flowers, and thus cross-pollination was accomplished. The pollen was transported by insects directly to the proper stigmatic surface with very little loss of material. The utilization of pollen as food by the insects and the assurance of successful cross-fertilization with a minimum of waste pollen had a survival value to both the insects and the plants. Cross-fertilization stimulated variation, and natural selection fostered and perfected those variations which favored insect pollination. The secretion of nectar, the bright color of flowers, and other characters that attract insects have evolved in this way. These interacting factors have resulted in strong evolutionary forces that have produced the exceedingly complex adaptations existing today between flowers and the insects that pollinate them. It would be out of place here to discuss in detail the thousands of intricate adaptations that evolved as a result of this association, but a few of the most important kinds of variation will be mentioned.

Plant Adaptations That Favor Insect Pollination.—The biological and evolutionary significance of cross-pollination has already been mentioned. Many of the adaptations of flowers are apparently the result of natural selection and perpetuation of variations in structure that favored or ensured cross-pollination. Some of these are independent of insect pollination and

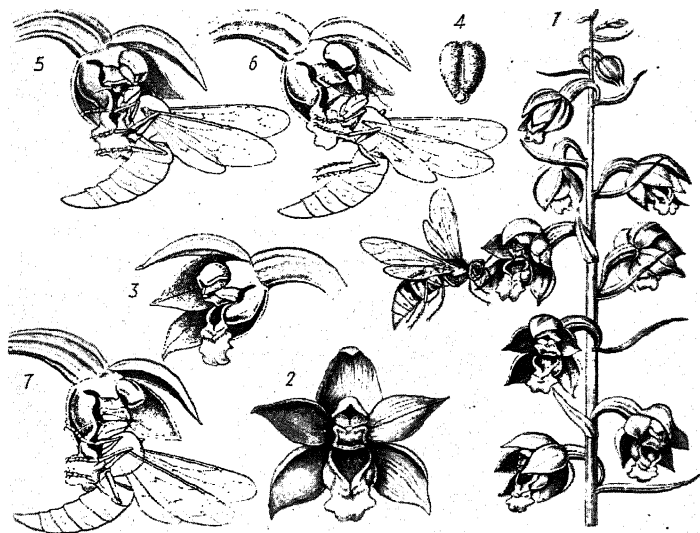


FIG. 19.—The transmission of pollinia by a wasp in orchid flowers (*Epipactis latifolia*). 1, the wasp alighting upon a flower; 2, the flower as seen from the front; 3, the same in side view; 4, a pair of pollinia with sticky pad at the base; 5, the wasp feeding upon the nectar and pressing its head against the pollinia; 6, the wasp leaving the flower with the pollinia adhering to its head; 7, the same insect visiting another flower, pressing the pollinia against the stigma, and effecting pollination. (After Kerner and Oliver.)

apply equally well to wind pollination, but many of them are also adaptations for insect pollination.

In many flowers, the stigma is receptive before its own pollen is ripe. In such cases, cross-pollination is usually accomplished before self-pollination is possible. When plants have only imperfect flowers or physiologically self-sterile flowers, self-pollination cannot occur, and cross-pollination is obligatory.

The pollen of many insect-pollinated plants is sticky or waxy, is not easily scattered by wind, and rarely reaches the stigma in

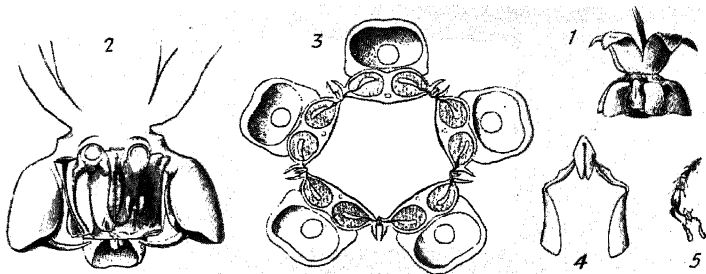


FIG. 20.—A flower of the milkweed (*Asclepias cornuti*) showing the pollinia and the mechanism by means of which they become attached to the feet of insects. 1, side view of flower; 2, the same magnified and with part of the anther removed to show the pollinia; 3, a cross section of a flower showing arrangement of the pollinia and the clips that become attached to the insects' legs; 4, a pair of pollinia and the closed clip mechanism; 5, a pair of pollinia attached to an insect's leg. (After Kerner and Oliver.)

the same flower; but it is transported readily by insects to other flowers. The specialized pollen masses, or pollinia, found in certain orchids, in milkweeds, and in other plants are structures

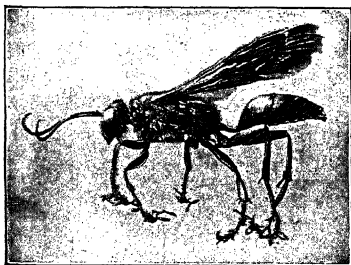


FIG. 21.—A wasp with pollinia of a milkweed attached to its legs by the clip mechanism shown in the preceding figure. (After Folsom and Wardle, by permission of P. Blakiston's Son & Company.)

that ensure cross-pollination by means of special adaptations for translocation by insects. The pollinia are sharply defined masses of sticky pollen specially adapted to insect transportation and borne so that wind dissemination does not occur. These adhere to some particular part of the insect's anatomy and are removed bodily from the stamens. When another flower is visited by the insect in search of nectar, the pollinia are pressed against the stigma, and cross-pollination is effected. In some cases, the pollinia adhere to the head (Fig. 19); in others, they catch on the legs or other parts of the insect (Figs. 20 and 21). However, in all cases, the arrange-

ment of the pollinia is such that they are removed from the stamens and are transported to the stigma of another flower. The pollinia are sharply defined masses of sticky pollen specially adapted to insect transportation and borne so that wind dissemination does not occur. These adhere to some particular part of the insect's anatomy and are removed bodily from the stamens. When another flower is visited by the insect in search of nectar, the pollinia are pressed against the stigma, and cross-pollination is effected. In some cases, the pollinia adhere to the head (Fig. 19); in others, they catch on the legs or other parts of the insect (Figs. 20 and 21). However, in all cases, the arrange-

ment prevents the pollen reaching the stigma of the flower in which it is produced but ensures that it will touch the stigma of the flower next visited by the insect.

The relative position of the style and stigma in insect-pollinated plants is usually such as to prevent self-pollination. As a general rule, the stigmatic surface is borne above the anthers so that the pollen will not drop upon it. In some flowers, there are explosive or tripping mechanisms that are set off by the insects in a way which ensures that their bodies become thoroughly dusted with pollen. The corollas of the flowers of some species of plants, especially among the orchids, appear to be so modified that pollination is effected by a single species of insect to the exclusion of all others. In other cases, several different species of insect may effectively pollinate the flowers of a single species of plant.

A further discussion of the innumerable modifications of floral structures concerned with insect pollination and cross-pollination is not possible here. The reader is referred to the extensive works of Darwin (1903), Knuth (1909), Kerner and Oliver (1895), Riley (1892), and others.

Insect Adaptations That Facilitate Insect Pollination.—So far in our discussion of insect pollination we have considered chiefly the modifications of floral structures. Similar adaptations exist also among the insects. Where there is mutual adaptation, it is not always possible to determine how much of the adaptation is the result of variation in the plants and how much is the result of insect variation. Because of the hybridization that occurs so frequently in plants as a result of cross-pollination, it seems reasonable to assume that the plants have been the more variable. However, hybridization and variation do occur in insects, and natural selection would operate whenever there are variations with a survival value. The most striking adaptation of insects for pollination are the specialized bristles that occur in a wide variety of forms and serve for collecting pollen (Fig. 22). These bristles are usually localized on that part of the insect's body which is most likely to come into contact with the stigma of the flowers that it usually pollinates. The suctorial mouth parts of insects that regularly feed on nectar present well-known adaptations. Some insects such as the Pronuba moth (*Pronuba spp.*) and the fig wasp (*Blastophaga psenes* L.) breed in the ovaries of

the plants that they pollinate. Associations of this kind usually result in a high degree of interdependence of the plants and their respective insects and involve highly adapted instincts.

The yucca plant is entirely dependent upon the moths for pollination. Its pollen is produced in sticky masses that preclude the possibility of wind dispersal. However, pollination is effectively accomplished by the pronuba moths whose breeding habits reveal striking adaptations of instinct. The female moth deposits her eggs in the ovary of the flower, and the young larvae



FIG. 22.—A honeybee, showing the many bristlelike setae with pollen grains adhering to them. One of the most important functions of the setae is the collection and transportation of pollen from flower to flower in which process the flowers are cross-pollinated. Fungus spores and bacteria adhere to the setae as readily as do pollen grains. (Cf. Fig. 220.)

are dependent upon the fertilized seeds for food. If the flowers were not pollinated, the seed would not develop, and the larvae would starve. With unerring instinct, the female moth collects pollen from one flower before ovipositing into the ovary of a second one. When oviposition is accomplished, she then stuffs the pollen into the funnel-shaped stigmatic opening (Fig. 23). Thus the insect not only makes fertilization and cross-pollination possible, but she also ensures a food supply for the young larvae. Although the larvae feed upon the developing seeds, they do not consume them all, a supply of seeds sufficient to maintain the species developing without injury. Without the aid of the moth no seed would develop.

An equally complicated adaptation is found in the relationship between the fig wasp and the Smyrna fig. The so-called "fig flower" is in reality a whole collection of flowers or an inflores-

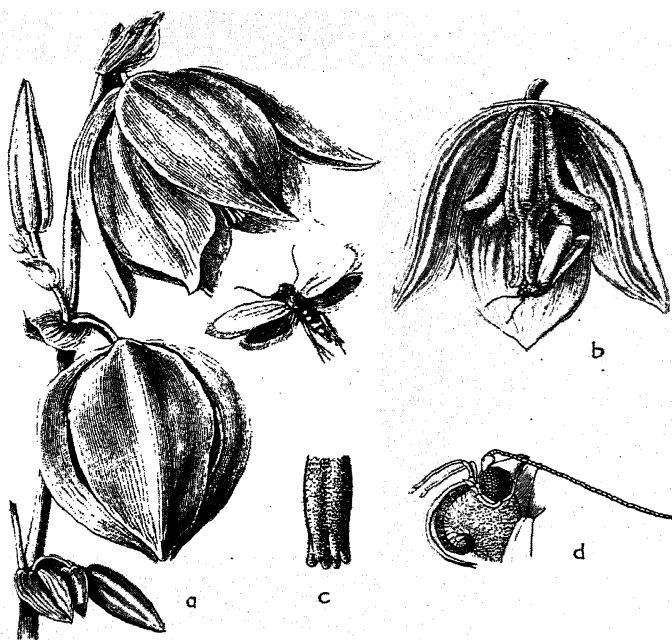


FIG. 23.—Insect pollination of yucca by the *Pronuba* moth. *a*, a sketch showing a *Pronuba* moth (*Pronuba yucasella*) and three yucca flowers in various stages of development; *b*, a yucca flower with three petals removed to show style and stamens. (The moth has oviposited in the ovary of the flower and is stuffing a mass of pollen, previously collected from another flower, into the funnel-shaped stigmatic surface); *c*, an enlarged view of the stigma; *d*, an enlarged view of the head of the female moth showing the mass of pollen grains held by the coiled maxillary palp that is specially adapted for this function. (After Kerner and Oliver.)

cence enclosed within a pear-shaped receptacle, a *synconium* (Fig. 24). The female of the fig wasp (*B. psenes* L.) enters the synconium through the eye and oviposits in the ovules of the young flowers. The eggs hatch, and the larvae develop in the ovary, which enlarges abnormally, forming the so-called "galls."

Following pupation the males emerge first and fertilize the females while they are still in the gall. The male dies without leaving the synconium, but the female emerges through the eye and in so doing becomes dusted with pollen. She then enters another fruit, and while in the act of oviposition she pollinates the pistillate flowers. In the edible fig, the styles of the pistillate flowers are so long that oviposition is not successful, and no wasps

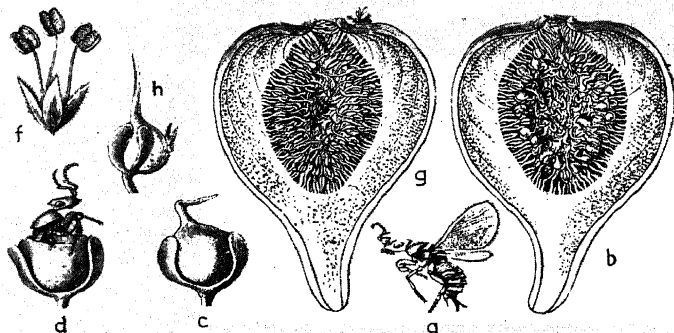


FIG. 24.—The pollination of the fig by the fig wasp (*Blastophaga psenes*). *a*, a fig wasp; *b*, a synconium of the caprifig showing the enlarged ovaries of the gall flowers in which the wasps develop; *c*, an enlarged view of the gall flowers of the caprifig showing the short style through which the eggs are inserted by the female fig wasp; *d*, a wasp emerging from a gall flower; *e*, a freshly emerged wasp which has become dusted with pollen from the male flowers (*f*) borne near the "eye" of the caprifig; *g*, a synconium of the edible fig the flowers of which will be pollinated by the wasp about to enter the eye; *h*, the female flower of the edible fig showing the long style which prevents successful oviposition. The insect thus pollinates the fruit of the edible species but is not able to breed in it. They breed in the caprifig which has the short styled flowers. (After Kerner and Oliver.)

develop in it. Pollination, however, is successful, and the fruits develop normally. The insects breed successfully in the inedible caprifigs, a supply of which is necessary in the cultivation of the edible varieties.

It is evident that in this relationship there is a high degree of interdependence between the fig plant and the fig wasp. Neither one can survive without the other. As will be described in Chap. VII, this insect is also an important vector of certain destructive diseases of the fig fruit.

5. INSECTS AND PLANTS THAT LIVE IN SYMBIOSIS

In the course of the long period of time that plants and insects have shared the available food and space on the earth's surface, many adjustments have been made. In numerous cases, certain species of plants and insects have become so thoroughly adapted to living together in close spatial relationship that the association has become relatively constant. This type of association is usually known as *symbiosis*.

We are interested here primarily in those microscopic plants (bacteria and fungi) which are known to live in symbiosis with insects. The existence of symbiosis between certain insects and microorganisms has been known for a long time, but the work of Paul Buchner and his students, summarized in his book "Tier und Pflanze in Symbiose" published in 1930, has shown us the astounding universality of the phenomenon and the high degree of development it has reached. Buchner's investigations are of great significance to the student of insects in their relation to plant diseases. They have shown that symbiosis with microorganisms is to be found in nearly all orders of insects and that in some groups, such as those feeding on plant sap and on wood, it is almost universal. In several cases, the symbiotic microorganisms are pathogenic to plants. Buchner's studies of the subject have yielded sufficient evidence to justify the conclusion that the interrelationships of insects and symbiotic microorganisms are as complex as, and of evolutionary significance equal to, those between insects and flowering plants. The almost universal occurrence of symbiosis between insects and microorganisms in general, coupled with the numerous striking cases involving plant pathogens, makes it imperative that we recognize the importance and significance of the phenomenon in the study of plant diseases. The subject is discussed in more detail in Chap. III.

6. INSECTS THAT DISSEMINATE PLANT PATHOGENS OR AID IN THE DEVELOPMENT OF PLANT DISEASES

This relationship is presented in Chap. IV.

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CHAPTER III

SYMBIOSIS BETWEEN INSECTS AND MICROORGANISMS AND ITS SIGNIFICANCE IN PLANT PATHOLOGY

Symbiosis is a subject of wide biological interest. The phenomenon is found in some form in almost all classes of plant and animal life and sometimes is essential to the survival of the species. Symbiosis is especially prevalent between insects and microorganisms, where the relationship often reaches a very high degree of development. It has been shown that a number of plant pathogenic microorganisms live symbiotically with their insect vectors, the relationship usually working to the mutual advantage of the associated species. Diseases caused by pathogens that live in symbiosis with insects are sometimes very complex in their ecological relationships. A satisfactory knowledge of the epiphytology of diseases of this kind is rarely possible without a thorough understanding of the symbiotic relationships of the pathogen and its insect vector. Therefore, symbiosis is obviously a subject that cannot be neglected in any comprehensive study of insect transmission of plant diseases.

Symbiosis, a term first used by De Bary (1879), is a broad one and includes all associations in which dissimilar organisms live together in close spatial relationship. It does not imply benefit or detriment to either party of the association. *Parasitism*, in which one organism derives benefit at the expense of the other, is a form of symbiosis usually termed *antagonistic symbiosis*. If the association is mutually beneficial to the associated organisms, it is designated *mutualistic symbiosis*, or *mutualism*. When there is little or no direct influence of one organism upon the other, the relationship is called *commensalism*.

It is not always possible to know accurately and completely how much and what kind of influence the organisms have upon one another. For this reason, it is often difficult to use these terms with accuracy. In much of the recent literature dealing with symbiosis between insects and microorganisms, the term

symbiosis has been used in a restricted sense to exclude parasitism and commensalism. Used in this sense, it has essentially the same meaning as mutualistic symbiosis. Nuttal (1923) defines symbiosis as "a condition of conjoint life that is more or less beneficial to the associated organisms or symbiotes," and Shull (1920) defines it as "the association of two species for their mutual benefit." Although there may be some question of the justification for restricting the term in this way, the restricted concept has become so well established that it may be very difficult to change it. However, in this discussion the use of the term in the restricted sense will be avoided as much as possible.

The term "symbionts" has been used very generally to designate organisms that live in a state of symbiosis, although some writers prefer to use "symbiotes," maintaining that it is the correct term (Meyer 1925). Symbiote seems to be the better word from the standpoint of etymology, and for this reason it will be used here. Some authors, in writing of symbiosis between insects and microorganisms, have used symbiont or symbiote to designate the microscopic member of the association, referring to the insect as the "host." Unfortunately, the latter term is used with different connotations in both entomology and plant pathology. In order to avoid confusion, the term "microsymbiote" will be used in this discussion in referring to the symbiotic microorganism. A good discussion of the origin, meaning, and usage of the above terms has been presented by Hertig, Taliaferro, and Schwartz (1937).

Perhaps the earliest recognized and best known example of symbiosis is that between the two microscopic plants found in lichens. The symbiotic relationship between the fungi and algae that make up the vegetative body of the lichens was first extensively studied by De Bary in 1879, and since then it has been the subject of numerous investigations which have shown that the benefits derived by the algae and fungi vary in different lichens. In some species, there is a mutual benefit; in other species the fungi are apparently parasitic on the algae, and instances have been reported wherein the algae are parasitic on the fungi.

Another well-known example of symbiosis between plants is that found in the root nodules on leguminous plants. The bacterial symbiote lives within the tissues of the root on which it causes the globular nodules characteristic of most leguminous

plants. Since the bacteria are able to fix atmospheric nitrogen, a source of nitrogen is provided for the legumes, which in turn furnish the bacteria with carbohydrates and other materials necessary for their growth.

It is well known also that the roots of most trees and many other perennial plants are invaded by symbiotic fungi. The fungus mycelium and the root tissues form specialized structures known as "mycorrhizae." These are either ectotrophic or endotrophic. In the former, the fungi are mostly on the surface of the root, surrounding it like a sleeve; those of the endotrophic mycorrhizae occur within the tissues, growing between the cortex cells. The physiological relationship between the fungi and the roots has been the subject of much study. The entire subject has been reviewed extensively by Rayner (1927) and by Hatch (1937). The latter concludes that the trees are dependent upon the fungi for the absorption of nutrients from infertile soils and that the fungi derive growth-promoting substances and in some cases simple carbohydrates from the trees.

Many lower forms of animal life characterized by a green or yellowish-green color have been known for a long time. It is now recognized that the color often is derived from symbiotic green algae living within the animal tissues. This condition is found in many Protozoa, Porifera, Coelenterata, Ctenophora, Platyhelminthes, Annelida, and Mollusca. The fresh-water forms are associated with green algae and are known as *Zoochlorella*. Those living in salt water harbor the yellow algae and are called *Zooxanthella*.

Symbiosis with microorganisms is very extensive among insects. Buchner (1930) has published an extensive review of the subject and has described a whole series of symbiotic associations of widely varying degrees of complexity. He is led to believe from his studies that this series of symbiotic relationships represents an evolutionary development, from a simple primitive type to the more complex associations, which parallels the evolution of the insects themselves. Thus in the Blattidae, where symbiosis is universal and of a similar nature in all species, it is believed that symbiosis arose in the most primitive ancestral forms and has been transmitted to the more recently evolved forms. In contrast to these, the Homoptera possess a variety of types of symbiosis in different species, a fact indicating that symbiotic rela-

tionships were established relatively late in this order and independently in the different groups within the order.

Buchner classifies the various types of symbiotic associations into two main groups: (1) *ectosymbiosis*, in which the symbiotic microorganism is found chiefly on the outside of the insect's body; and (2) *endosymbiosis*, in which the microorganisms are harbored within the body of the insect. Endosymbiosis is further divided into a progressive series of increasingly complex types on the basis of the localization of the microsymbiote within the insect body. These subclasses are as follows:

a. The microsymbiote develops free in the lumen of the intestinal tract or in intestinal caeca.

b. The microsymbiote develops within the epithelial cells of the intestinal tract.

c. The microsymbiote develops in the region of the mesoderm but retains its connection with the epithelium.

d. The microsymbiote develops within special cells (mycetocytes) or special tissues (mycetomes) without any connection with the intestinal tract.

Ectosymbiosis is considered the most primitive type followed by the simpler type of endosymbiosis in which the microsymbiote is found in the lumen of the intestine or in intestinal caeca. From this simple type of endosymbiosis, more complex types of endocellular symbiosis have evolved. It is believed that the microorganisms entered the epithelial cells, finally invaded the mesoderm, and, in the most highly developed types, became localized in mycetocytes or mycetomes, having lost all connection with the intestinal organs. A better appreciation of this classification may be had from the consideration of representative examples.

Ectosymbiosis.—Perhaps the most widely known example of ectosymbiosis is that between the ambrosia beetles and the ambrosia fungi. The ambrosia beetles are timber-boring Scolytidae that breed in the sapwood of dying trees. The galleries are of various types, but the walls are always stained a dark color by the growth of a characteristic fungus that is universally present. The fungus is introduced by the beetles into the newly formed gallery where it grows to the exclusion of other fungi. The ambrosia fungus fruits by forming masses of glistening white spores on the walls of the larval chambers. The developing larvae feed upon the fungus spores, deriving most of

their food from this source. When the new brood of beetles emerges, each beetle carries with it spores of the fungus to be used in starting a new culture in the galleries established in suitable trees. The somewhat similar association of bark beetles and fungi as illustrated in the Dutch elm disease and in the blue stain of conifers (see Chap. VII) is another example of ectosymbiosis.

The fungus-cultivating ants and termites and their fungus gardens are also examples of ectosymbiosis. Both these groups of insects cultivate fungi in their domatia on specially prepared beds of plant tissue. The growing fungus is utilized as food by the developing young; and when a new colony is established, enough of the fungus is carried along to start a new culture.

Endosymbiosis.—*a.* The simplest type of endosymbiosis is that in which the microsymbiote develops in the lumen of the intestinal tract of the insect without penetrating the epithelial cells. Excellent examples of this relationship are found among the Diptera, many species of which harbor bacteria that aid the insects in the digestion of the tissues on which they feed. Within this group is found a series of associations of increasing complexity, varying from the relatively simple type between bacteria and the housefly (*Musca domestica* L.), the blowfly (*Calliophora vomitoria* L.), and the seed-corn maggot (*Hyalemyia ciliatula* Rond.) in which there are no special caeca for harboring the microsymbiote, to the more complicated type between the olive fly (*Dacus oleae* Rossi) and its microsymbiote in which highly specialized caeca, or "fermentation chambers," are provided for harboring the bacteria and ensuring contamination of the eggs (see Chap. VI).

b. A good example of a microsymbiote localized in the epithelial cells of the insect is afforded by the yeast associated with the flour beetle (*Sitodrepa panicea* L.). Certain cells (mycetocytes) along the walls of the intestinal caeca of this insect are filled with actively growing yeast cells. The yeasts, which aid the beetles in digesting starchy food materials, are also found in the intestinal lumen and in special caeca from which the eggs are surface-contaminated when deposited.

c. In the weevils, represented by *Hylobius abietis* L., the microsymbiotes are found within groups of cells (mycetomes) adhering closely to the walls of the intestinal tract but having no direct

connection with the lumen. These are often so situated that they resemble the caeca, or fermentation chambers, that harbor the microsymbiotes in the more primitive forms described above. This resemblance has suggested that they may have originated as a modification of the fermentation chambers, the walls of which have been transformed into mycetomes. Although the function of these mycetomes is not conclusively known, they are supposed to be concerned in digestion of the wood consumed by the weevils.

d. The most highly developed type in this series is represented by those insects which harbor microsymbiotes in specialized groups of cells, or mycetomes, located deep in the body cavity and having no connection with the intestines. This situation is especially prevalent in those sucking insects which commonly feed on the sap of plants, such as aphids, scale insects, and leaf hoppers. The so-called "pseudovitellus" of the aphids is the classic example of a mycetome of this type. This organ, consisting of a mass of large fatlike cells found in the abdomen of aphids, was described by Huxley in 1858, but it was not until 1910 that its true nature was discovered independently by Pierantoni and Sulc. In this association, the microsymbiotes serve the insect by absorbing waste products, such as urates, and the insects provide the microsymbiotes with food and protection.

When insects and microorganisms live in symbiosis, some provision is necessary to ensure constant association of both symbiotes. Obviously, the greatest necessity is to ensure the transmission of the microsymbiote from one to the next generation of the insect. This is accomplished in a variety of ways. Where the symbiotic microorganism is cultivated outside the body, the insects always carry the microsymbiote with them when establishing a new colony. In many cases, the transportation appears to be a matter of chance, the microorganisms adhering to the surface of the insect's body or being carried in the contents of the intestinal tract and being passed out with the excreta in a viable condition. This is the method used by the bark beetles and many of the ambrosia beetles. The females of some tropical ambrosia beetles are said to carry spores and mycelium of the ambrosia fungus between special brushlike structures on the front part of the head (Strohmeyer 1911). In the case of the fungus-cultivating ants, the female stores in her infrabuccal pouch a

mass of the fungus which is used as "spawn" in starting a new fungus garden.

More highly specialized devices for transmission of the micro-symbiote are found in insects with endosymbiosis. Here there appears to be a series of increasingly complex arrangements. The simplest arrangement is that found in certain dipterous insects in which the reinfection of larvae is left entirely to chance, the eggs usually being deposited in places where the microorganisms are abundant. In certain other insects, the anal and the genital openings are so close together that the eggs become surface-contaminated from the excreta at the time of oviposition, no special organs for ensuring contamination being necessary. Next in order of complexity are those in which there are anatomical modifications of the female body involving special organs that function to ensure contamination of the egg. These organs differ considerably in different species, but all involve the accumulation of the microsymbiote in some sort of diverticulum located near the genital opening in a way that ensures the contamination of the eggs at the time of oviposition. In some species, contamination depends upon the normal pressure of the egg as it passes through the oviduct. In others, the diverticulum is equipped with special muscles that force the microorganism out onto the egg surface. Some of these diverticula arise from the anal passage, some are intersegmental in origin, and in certain species the *bursa copulatrix* is used for this purpose.

In the majority of cases, the young larva comes in contact with the microsymbiote only after the egg has hatched and the larva has had an opportunity to ingest the microorganism from the shell surface. But in some insects, as in *Dacus oleae*, the bacteria penetrate the egg through the micropyle, and the larva is internally contaminated before the egg hatches.

In those insects in which the intracellular symbiotes are located in mycetocytes or mycetomes contained in the body cavity, the eggs generally become infected internally before oviposition, the microsymbiote entering the immature eggs through the nurse cells. Some of the scale insects offer good examples of this kind of transmission. Mansour (1934a) has described a different process found in three beetles (*Rhizopertha dominica* F., *Sinoxylon ceratoniae* L., and *Bostrychophilites* Marsh.) in which the microsymbiotes invade the testis lobes, multiply and mix with the

sperms, and during copulation pass into the *bursa copulatrix* of the female. From here they penetrate through the micropyles of the fully formed eggs during oviposition.

In some of the parthenogenetic insects, reproducing viviparously, the young embryo is infected before birth. This is the rule among the aphids. In the Pupipara and in certain other Diptera (Glossina) that reproduce viviparously, the young larvae are contaminated through the so-called "milk glands" which nourish them before birth.

THE NATURE OF THE SYMBIOSIS BETWEEN INSECTS AND MICROORGANISMS

Most, if not all, the symbiotic associations between insects and microorganisms rest upon a physiologic basis and probably should be classed as mutualistic symbiosis. The associations, however, are very complex, and in only a very few cases is there enough known about the physiology of the symbiotes to justify a final conclusion. In reality, it is practically impossible to draw a sharp line between parasitism and mutualism in most instances of symbiosis of insects with microorganisms. Well-defined cases of insects being parasitized by microorganisms are known, and there are equally well-defined examples of mutualistic symbiosis; but between these two there is a whole series of intergrading types that permits no sharp separation. Perfect mutualistic symbiosis might be considered as an association in which both symbiotes are so completely adapted to a common life that neither one can survive in the absence of the other. This condition has been called *obligate symbiosis*. Such relationships are extremely rare. There are, however, numerous associations in which both symbiotes are constantly associated in nature and in which the survival of one of the symbiotes is dependent upon the presence of the other. In these cases, it usually can be demonstrated experimentally that the other symbiote may survive alone under proper conditions, although it may never be found alone in nature. Beginning with these highly specialized associations, we find all degrees of interdependence down to very casual associations that are by no means fixed or constant.

Many students of symbiosis have concluded that mutualistic symbiosis arose through an intermediate stage of parasitism

on the part of one of the associated organisms. According to this theory, the parasitized member acquired an increasing degree of immunity which, after a period of time, resulted in mutual adaptation to a common life. If this theory is correct, the microsymbiotes were originally parasitic on the insects, the latter having acquired immunity sufficient to establish a fairly constant equilibrium.

Buchner (1930) expresses the opinion that, in all cases of symbiosis between insects and microorganisms, it is the insect which dominates the situation. The microsymbiote is provided with food and shelter but in all other respects is dominated by the insect that derives the greater benefit. Buchner, therefore, speaks of the association as a "master-servant" relationship.

The frequency with which symbiosis occurs in nature has led some biologists to suggest that the phenomenon has a general and fundamental significance in all forms of life. As a matter of interest the hypothesis formulated by Portier (1918) should be mentioned, although it has been generally discredited. Portier held all higher forms of life to be the results of symbiosis. He recognized two main groups of living organisms, separated on the basis of the occurrence of intracellular symbiosis: the *autotrophic* forms which included only the simplest unicellular organisms such as bacteria, and the *heterotrophic* forms which included all higher plants and animals, the tissues of which, according to Portier, constantly and universally contained intracellular symbiotic microorganisms. Some of these symbiotes, he claimed, could be isolated and grown in artificial culture while others had become so thoroughly adapted that they could not be isolated and cultured. He interpreted the latter as identical with the mitochondria or other plastids. These intracellular symbiotes, according to Portier, play a very important role in all biologic processes such as sexual fertilization and disease resistance and even in the origin of species. The theory was given some support by the Italian biologist Pierantoni and has been elaborated by Wallin (1927) in his book "Symbiogenesis and the Origin of Species."

In the consideration of mutualistic symbiosis, one is interested in knowing the physiological interrelationships and the relative amount of benefit derived by each party of the association. However, so little is known of the physiology of either the

insects or the symbiotic microorganisms that not much accurate information is available. Uvarov (1928) has summarized the literature on the nutrition and metabolism of insects up through 1927, emphasizing the great lack of accurate and specific information on the subject. For our purposes, it will be necessary to discuss only a few of the facts and theories that have come from some of the more important investigations.

Apparently the benefits derived by the insects from the association are largely a matter of nutrition. Several investigations have shown that symbiotic microorganisms aid insects in the digestion of their food. The microsymbiotes apparently produce enzymes, vitamins, or other substances that the insects themselves cannot produce. One of the more clear-cut cases of this type is that of the wood-eating termites and protozoa investigated by Cleveland (1923 to 1934) who demonstrated that, for the digestion of cellulose, these termites are dependent upon protozoa found in an enlargement of the hind intestine. When the termites were defaunated and reared in the absence of the protozoa, they were unable to digest cellulose.

In support of the theory that the microsymbiotes are largely concerned with nutrition, Buchner (1930) points out that the insects which most universally harbor microsymbiotes can be grouped into the following four general categories on the basis of the type of food utilized:

1. Those that utilize food rich in carbohydrates (especially cellulose) and poor in nitrogen.
2. Those that suck the sap of plants.
3. Those that feed on blood of vertebrates.
4. Those that feed on horny substances, hair, feathers, etc.

Baumberger (1919) made extensive experiments with *Drosophila* and concluded that symbiotic yeasts were necessary for the normal development of the larvae. He concluded that the larvae utilized the yeast as food and that the chief role of the yeast was in the synthesis of some necessary food substance or the concentration of available nitrogen. He held that the nitrogen content of the plant tissues was not sufficient to allow the completion of the life cycle in the normal length of time. He showed that the edible portion of banana tissue contained about 1.3 per cent protein while the yeast cells contained about 11 per

cent. These general conclusions conform with those reached by Loeb and Northrup (1916) and Northrup (1917).

Leach (1926, 1931) and Huff (1928) have shown that the bacteria associated with the seed-corn maggot, the vector of potato blackleg and other soft rots, aid the insect in the digestion of the plant tissues on which it feeds. Sterile maggots reared from surface-sterilized eggs do not develop normally when fed on sterile plant tissue such as a potato plug or steamed bean seeds, but if the bacteria are present they grow well and pupate normally. They will grow and pupate also on tissues previously decayed by the bacteria and later sterilized by heat or on sterile germinating beans or pea sprouts in which the stored foods are being translocated into the growing shoots. These facts indicate that the chief function of the bacteria is to digest the plant tissues and make them available as food for the larvae.

It has been shown by numerous investigators that many insects feeding on decayed wood derive their nourishment primarily from the fungi found in the wood. The nitrogen content of the wood appears to be the limiting factor in the nourishment of many wood-eating insects. Since fungi have a higher nitrogen content than wood, those insects feeding on decayed wood are able to complete their life cycle in a shorter period of time than those feeding on sound wood.

Buchner (1928) maintains that the intracellular symbiotes of wood-eating beetles digest cellulose that the insects alone could not digest. This interpretation is questioned, however, by Mansour (1934b) and Mansour and Mansour-Bek (1934) who claim that the relationship is not one of mutualistic symbiosis but is to be interpreted as commensalism. Müller (1934) interprets the relationship as a very weak regular hereditary parasitism.

Uvarov (1928) and Koch (1933) conclude that the micro-symbiotes in many instances are utilized as food by the insects and serve as a source of vitamins that the insects require but cannot synthesize. Bacot and Harden (1922) investigated the role of yeast in the nutrition of *Drosophila* and concluded that the insect requires vitamin B for complete development and that the vitamin is normally obtained from the yeast. These conclusions have been confirmed by Van't Hoog (1935), who was able to grow the insects successfully in synthetic medium,

concluding that in nature the yeasts commonly supply one or more of the B vitamins and some undetermined substance in the unsaponifiable fraction of fats.

It has been suggested that those microsymbiotes which are associated with the Malpighian tubules play a part in decomposing and disposing of the waste products of the insect's metabolism. Some suggest, without supporting evidence, that the microsymbiotes fix atmospheric nitrogen for certain insects that feed on materials low in nitrogen content (Cleveland 1925a). Others believe that the microsymbiotes found in mycetomes of the Homoptera are concerned in making available the food materials stored in these structures for the nourishment of the developing ova. It is even suggested that they may be concerned in the initiation of embryonic development in those insects that reproduce parthenogenetically.

The fungi associated with the bark beetles develop extensively in the bark surrounding the brood galleries and modify greatly the microenvironment by increasing the moisture content of the inner bark and causing the outer bark to separate from the wood. Leach, Orr, and Christensen (1934) considered this ecological influence of decided value to the insect. Person (1931) has presented evidence to show that the yeasts associated with the bark beetles ferment the sap of infested trees and produce a distinct odor that attracts other beetles, making it possible for the insects to attack the trees gregariously. This obviously would be of definite value to the insects because individual insects are rarely able to establish themselves in normal, vigorous trees.

In spite of the scarcity of experimental evidence, there are several obvious ways in which the microsymbiote profits by the association. In the first place, the microorganisms are assured a constant food supply and effective protection against such unfavorable environmental factors as excessive heat, light, and desiccation. If the microorganisms is not an obligate symbiote, it frequently is associated with the same food substance utilized by the insect. Organisms of this kind usually depend upon the insect for dissemination and transportation to the source of food. Also, if the microsymbiote happens to be a plant pathogen, it may be dependent upon the insect for ingress into the plant tissues (*cf.* the Dutch elm disease, bacterial wilt of cucurbits, potato blackleg, etc.).

There is obviously much variation in the functions of the different symbiotes and the benefits derived by each; yet the evidence seems to indicate more than a fortuitous relationship. It would be difficult to conceive of such complicated and specialized associations as arising through chance and having no biological value. The associations are certainly the result of a gradual evolutionary development and have persisted because of some survival value, although the survival value may have been based on any one of many different functions. There is no reason to expect that the functions and benefits should be the same in all cases.

Symbiosis gives rise not only to physiological adaptations but to structural modifications as well. There have been numerous anatomical adaptations on the part of the insect for harboring and transporting the microsymbiote and for ensuring its transmission from generation to generation. Among these are the various intestinal caeca, mycetocytes, and mycetomes, examples of which have already been described.

The structural modifications on the part of the microorganisms are not so evident. The fungi cultivated by the leaf-cutting ants develop peculiar club-shaped hyphal tips that are eaten by the ants. These structures are called *bromatia* (Fig. 34) and are said by Wheeler (1923) and others to be the result of some special form of cultivation by the ants. As a rule, the intracellular microsymbiotes are very simple in structure and show no special morphological adaptations. However, Buchner (1930) believes that they have undergone a reduction in both form and function as a result of their symbiotic mode of life. According to this view, the simplicity of form is in itself a structural adaptation to symbiotic life.

The significance of the phenomena of symbiosis in a study of the relation of insects to the spread and development of disease is quite obvious from the preceding discussion. In many cases of symbiosis with insects, the microsymbiote has proven to be a plant pathogen. With increasing frequency, new cases are being discovered in which insect vectors of important plant diseases maintain some degree of mutualistic symbiosis with the pathogens. The recognition of these interrelations and a thorough understanding of their nature are necessary for intelligently combating the diseases.

The investigations by Carter (1933) of the green-spotting disease of pineapples reveal another way in which the micro-symbiotes of insects may be significant in the field of plant pathology. Carter has shown that green spotting is caused by a phytotoxic secretion of the mealy bug and that the toxic secretion is closely correlated with the presence of a rod-shaped symbiote localized in the mycetome of the insect. The increasing recognition of destructive plant diseases caused by toxicogenic insects and the possibility of the toxic secretions being produced by the micro-symbiote make it imperative that the phenomena of symbiosis between insects and microorganisms be more thoroughly investigated from this viewpoint.

The almost universal occurrence of well-developed mycetomes and micro-symbiotes in those insects which are most effective as vectors of virus diseases has suggested the possibility that they may be in some way concerned (Rambousek 1929). However, no positive evidence of a relationship has been presented, and the more recent advances in the study of virus disease give little support to the idea.

SELECTED EXAMPLES OF SYMBIOSIS BETWEEN INSECTS AND MICROORGANISMS

Ambrosia Beetles.—Schmidberger in 1836 was probably the first to recognize and describe the association of the beetles with the so-called "ambrosia." He observed that the beetle larvae fed upon a glistening white substance. He did not understand the fungus nature of the substance, interpreted it as a product of the exuding plant sap, and applied the term "ambrosia" to it. Three years later, Ratsburg (1839) confirmed Schmidberger's observations and suggested that the ambrosia was the product of a mixture of plant sap and insect spittle. The fungus nature of the ambrosia was first recognized in 1844 by Thomas Hartig who described the fungus associated with *Xyleborus (Bostrichus) dispar* Fabr. in *Alnus cordata* and named it *Monilia candida*. Hartig, being a believer in heterogenesis, thought the fungus arose from the wood cells acted upon by a stimulating substance secreted by the beetles.

Despite the interesting nature of this association of fungi and beetles, there has been remarkably little study of it. Hubbard in 1897 made what is probably the most extensive study of this

group of insects but made only casual observations of the associated fungi. He concluded, however, that there were more than one ambrosia fungus and that only the most closely related species of beetles have the same fungus for food. Unfortunately, there has been no comprehensive study of the ambrosia fungi. Many casual references and brief descriptions are found in the literature, but no systematic study of the ambrosia fungi has been made. Until quite recently, the name used by Hartig, *Monilia candida*, was the only one applied to this group of fungi. We are indebted to Neger (1908 to 1911) and Schneider-Orelli (1911, 1913), who worked chiefly with species of *Xyloterus* and *Xyleborus*, for the most thorough studies that have been made on any of the ambrosia associations. These investigators have described accurately the fungi associated with the beetles studied and have shown how the fungus is cultivated by the beetles and how it is transmitted from one generation to the next.

The true ambrosia beetles belong to the family Scolytidae. They infest a wide variety of trees throughout the tropic and temperate areas of the world and for the most part are found in the wood of trees weakened by some unfavorable condition. They are rarely found in vigorous, rapidly growing trees or in dead ones, although there are some exceptions. They bore deep into the sapwood, each species making its own characteristic brood tunnels. In all cases, the walls are stained a dark color by the associated ambrosia fungus. The tunnels and breeding habits are of two general types. The beetles of one group of genera are semisocial in nature, rearing their young in large communal galleries and sealing dead members of the colony in special death chambers. In the other group, each larva develops in its own separate larval chamber, excavating it as it grows (Fig. 25). In both groups, the chief food of the developing larvae consists of the spores of the ambrosia that fruit in white masses on the surface of the larval chambers. According to Hubbard (1897), the ambrosia fungi associated with these two groups of beetles also are different as indicated by the method of spore formation.

The ambrosia fungi invade the wood for only a short distance from the tunnels and apparently do not cause a decay of the wood although the cells adjacent to the tunnels are stained dark brown or black. The fungi live chiefly on the contents of the

cells of the sapwood and do not destroy the cell walls. The value of infested wood is decreased when used for structural purposes, but otherwise the wood is not greatly injured.

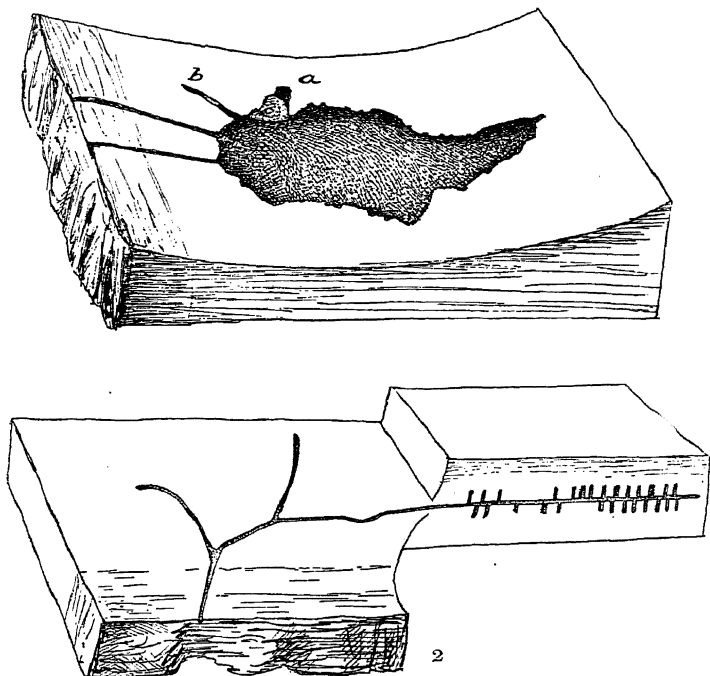


FIG. 25.—The two general types of ambrosia-beetle tunnels. 1, the communal gallery of *Xyleborus xylographus* in hickory wood; *a* and *b*, death chambers, or special portions of the tunnels in which dead individuals are sealed off. The ambrosia fungus grows and sporulates over the surface of the entire communal gallery. 2, the tunnel of *Monarthrum mali*, the type characterized by individual larval galleries. The ambrosia fungus fruits on the surface of the larval galleries. There appear to be two general types of ambrosia fungi corresponding to the two types of tunnel. (After Hubbard.)

In those genera in which the young are reared in a common gallery, the larvae have mouth parts not suited for chewing wood, and they apparently feed entirely on the fungus. The larvae that develop in individual chambers have strong mandibles that are used to bore out the chamber. In doing this, they consume some wood in addition to the fungus. Although

there is no experimental evidence, it is generally assumed that the ambrosia fungi, by concentrating the nitrogenous elements, serve as a supplement to the wood and provide the insects with a more suitable and efficient diet. Other fungi and yeast are often found in the tunnels, but they are suppressed by the beetles so that the ambrosia fungus grows as if in pure culture. As soon as the tunnels are deserted by the beetles, the ambrosia fungus is immediately overgrown by secondary fungi.

According to Schneider-Orelli, the ambrosia fungus of *Xyleborus dispar* is transmitted to successive generations in the form of spores in the crop of the female beetle who regurgitates them to start a culture in the new tunnel. Neger, however, thinks the spores are passed through the insect's body and survive in a viable condition in pellets of excrement. Schneider-Orelli states that the spores taken directly from the tunnels of *X. dispar* do not germinate, but if recovered from the crop of the female beetle they germinate readily. However, this is not true for all ambrosia fungi. Strohmeyer (1911) has described several species of ambrosia beetles from specimens collected in the tropics in which the female beetles have special chitinous bristles on the front part of the head in which spores and mycelium of the ambrosia fungi are always found. He has interpreted these structures as special organs for the transportation of spores and mycelium of the fungus to be used in establishing cultures in the new brood chambers.

Taxonomic studies of the ambrosia fungi are conspicuous by their absence. Neger (1908, 1909) expressed the opinion that the ambrosia fungus of *Xyleborus* is an Endomycete, but neither he nor Schneider-Orelli (1913) reached a final conclusion concerning the identity of the fungus. The fruiting structures of these fungi as they occur in nature are relatively simple, and very few of them have been studied in artificial culture. Trotter (1934) recently reported the study of an ambrosia fungus associated with a beetle of the genus *Xyleborus* found in the wood of *Brownea grandiceps* Jacq. in Ceylon. He observed the "ambrosia" in material imported into Italy from Ceylon, grew the fungus in several hanging drop cultures, and observed the manner of fructification. In addition to the usual Monilia-like growth, he observed a second layer of mycelium superimposed upon it and forming an abundance of fusiform, hyaline

spores. On the basis of limited observations, he concluded that the superimposed fungus was a second spore form of the first. For this pleomorphic ambrosia fungus, he established a new genus *Ambrosiomyces* and named it *A. zeylanicus*.

Leach, Hodson, Christensen, and Chilton (1940) have recently reported observations on two species of ambrosia beetles (*Trypodendron betulae* Sev. and *T. retusus* Lec.) found in white birch (*Betula papyrifera*) and in aspen (*Populus tremuloides*), respec-



FIG. 26.—A, a section through a portion of a brood gallery of *Trypodendron retusus* in aspen wood showing the main tunnel blackened by growth of the ambrosia fungus and several larval chambers arising vertically at right angles from the main tunnel. The eggs are deposited in small niches in the upper and lower surfaces of the main tunnel. When they hatch, the larvae bore their tunnels as they grow. The white fruiting layer of the fungus may be seen on the walls of the larval chambers. Two larvae are shown in their chambers. B, an enlarged view of a small section of an egg gallery and a single larval chamber showing the white mass of ambrosia fungus consisting largely of spores.

tively. These closely related species of ambrosia beetles are found only in dying trees. They are unable to establish themselves in living trees, and dead ones are not attractive to them probably because of low moisture content. The tunnels are more abundant near the base of the trees, their frequency gradually diminishing toward the top. The upper limit of infestation coincides very closely with the lower limit of the living bark. The life histories and breeding habits of the two species are very similar.

Both species form brood galleries in which the larvae are reared in individual larval cradles (Fig. 26). In those of *T.*

retusus, there is a single entrance tunnel that extends into the wood for a short distance ($\frac{1}{4}$ to $\frac{1}{2}$ inch) and then branches to form two tunnels passing in opposite directions horizontally through the sapwood nearly parallel to the surface. The eggs are deposited in niches on the upper and lower surfaces of the galleries. The galleries of *T. betulae* are very similar to those of *T. retusus*, but they penetrate more deeply into the heartwood.

There is only one brood of the beetles annually. Infestation takes place in May, and the new brood matures in late summer and leaves the trees shortly thereafter. The beetles apparently do not overwinter in the trees, but it is not known where and how they hibernate.

The ambrosia fungi associated with these two beetles also are very similar and are in all probability merely strains of the same species. The ambrosia fungus is obviously introduced into the wood by the beetles and may be observed growing on the walls of the tunnels within a few days after the beetles enter the tree. It appears first as a glistening white or cream-colored mycelial growth but rapidly becomes darker, staining the walls a dark-brown color that is almost black. The walls are thoroughly invaded by the brown mycelium, but the fungus does not sporulate extensively in the main gallery. The female beetles deposit their eggs in niches, and each egg is covered with a pad of frass that is always permeated with mycelium of the ambrosia fungus. When platings are made from the frass that covers the eggs, cultures of bacteria, yeasts, and other contaminating fungi are often found, but the mycelium of the ambrosia fungus predominates. In a way not clearly understood, the beetles are able to suppress the development of all extraneous fungi so that the ambrosia fungus appears to be growing in pure culture. However, if the beetles are removed from the tunnels, the ambrosia fungus is completely overgrown by other fungi.

As the eggs hatch, the young larvae bore out their own cradles, enlarging them as they increase in size. The larvae are oriented with their heads away from the main tunnel, and as they feed upon the wood they enlarge their cradles. The frass is pushed out into the main tunnel, from which it is pushed to the surface by the adult beetles. Almost as soon as the larvae begin to enlarge the egg niche into a larval cradle, the ambrosia fungus begins to fruit on the surface of the walls of the cradle in white,

glistening masses (Fig. 26*B*). The hyaline unicellular spores are produced on short, erect sporophores arranged in palisade layers over the surface of the wood (Fig. 27). These spores are eaten by the larvae as they enlarge the cradle, but new layers of spores are repeatedly formed.

Prior to pupation, the larvae reverse their usual position in the cradle and face the main gallery, occupying this position during metamorphosis; on maturity, the new beetles eat their way through the plug composed of frass on which the fungus is usually fruiting (Fig. 28). The beetles thus have every oppor-

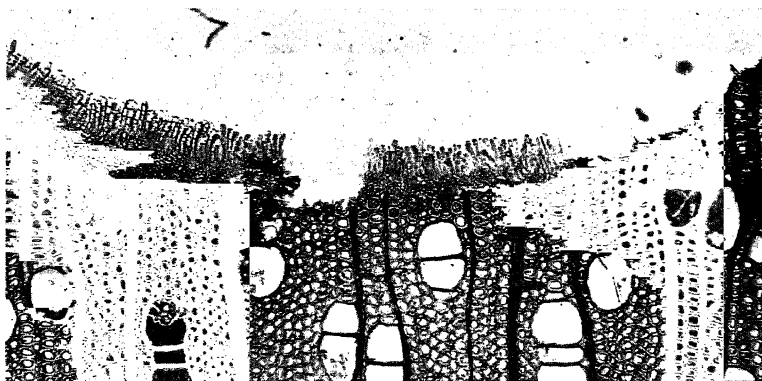


FIG. 27.—A cross section of the wood forming the wall of a larval chamber of *Trypodendron betuli* showing the palisade layer of sporophores of the ambrosia fungus. Approx. 100 X.

tunity of becoming contaminated with the ambrosia fungus both internally and externally before they emerge. The fungus does not survive metamorphosis within the body of the pupae. The fate of the fungus after emergence and during hibernation has not been determined.

The spores of the ambrosia fungus germinate very erratically on artificial media, those of the fungus associated with *T. betulae* germinating more readily than the others. Spores produced in artificial culture germinate more readily than those taken directly from the tunnels. Both strains of the fungus have been obtained in pure culture where only minor differences could be observed. The mycelium is hyaline at first, becoming brown with age and staining the medium brown. At first the fungi sporulate very



FIG. 28.—Two pupae of *Trypodendron retusus* with heads toward the main gallery. Note the white masses of ambrosia fungus growing on the mass of frass separating the pupal chamber from the main tunnel. Approx. 8 \times .

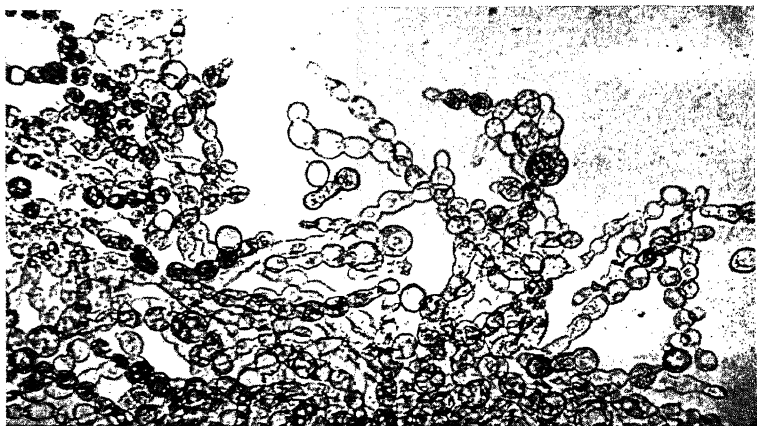


FIG. 29.—The ambrosia fungus of *Trypodendron betuli* showing monilioid type of fructification in artificial culture.

poorly in artificial culture, forming only imperfect monilioid spores that tend to remain attached and to bud *in situ*. White patches and sectors, both of which produced large numbers of hyaline, erect sporophores, later arose on the brown mycelial culture. When the patches or sectors were subcultured, a culture that sporulated abundantly was obtained. The spores formed in culture are hyaline with thin walls and are ovoid to round. They tend to be more spherical than the spores formed in the tunnels (Fig. 29). They range from 6 to 17 microns in length and from 6 to 14 microns in width with average dimensions of 11.38 by 10.09 microns. The spores are cut off basipetally, and the growth of the sporophore is indeterminate.

The fungi fall within the range of the genus *Monilia*; and, pending more extensive taxonomic studies of the ambrosia fungi, they are considered to belong to the species *Monilia candida* Hartig.

Termites and Fungi.—The termites, although considered a primitive group of insects, are equal to the ants and the bees in the complexity of their social organization. Their extensive caste system, their fungus cultivation, and other aspects of their social life have been so well described by Wheeler (1907, 1923) and Kofoed *et al.* (1934) that only the more significant aspects of their associations with fungi and other microorganisms will be included in the following discussion.

Termites are a primitive, although ancient, group of insects closely related to the cockroaches. They live in highly organized colonies and have a complicated caste system including workers, soldiers, and reproductives (Fig. 30). As a group, they feed largely on cellulose and fungi.

The termites fall into two large groups, based on their food habits. One group feeds primarily on wood and associated fungi, and the other feeds largely on fungi that are cultivated in special fungus gardens in their domatia. The wood-eating termites are often extremely destructive to buildings and other wooden structures.

The fungus-cultivating termites are confined chiefly to Asia, Africa, Australia, and various islands of the Old World. They live in large nests, or termitaria, which may be built underground, or in variously shaped, mud-covered structures above-ground (Fig. 31). The fungi are cultivated on the excreta of

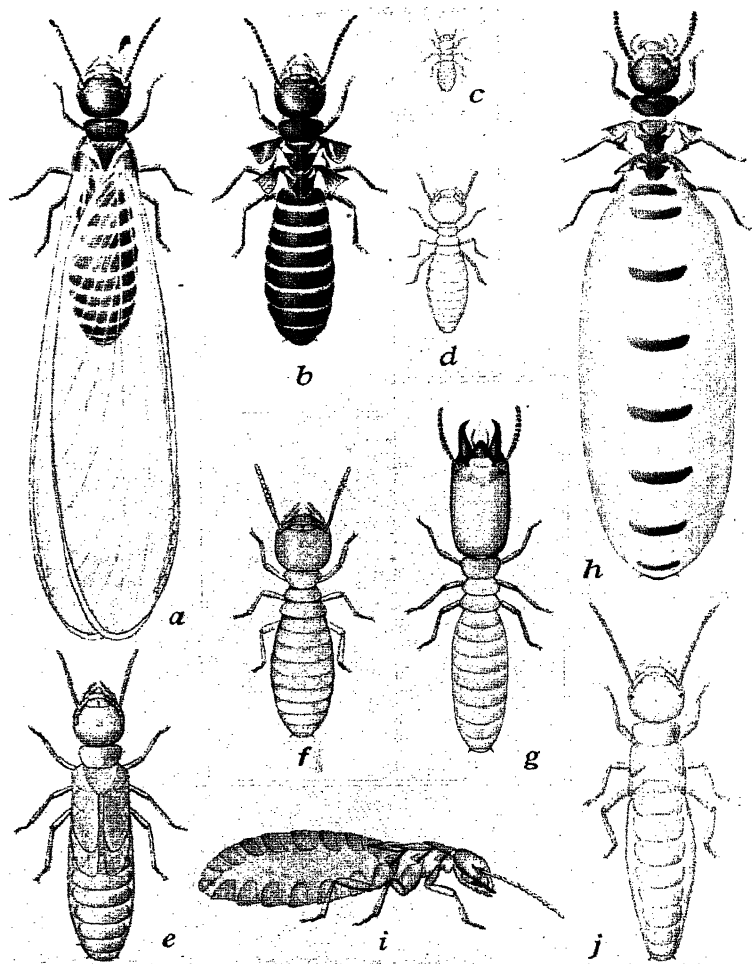


FIG. 30.—The different castes and nymphal stages of the western subterranean termite (*Reticulitermes hesperus* Banks). *a*, winged reproductive; *b*, dealated reproductive; *c*, a very young nymph, caste not yet evident; *d*, third-instar nymph, caste not evident; *e*, late nymph of reproductive caste; *f*, mature worker caste; *g*, mature soldier caste; *h*, mature breeding queen; *i*, short-winged, brachypterous, or second-form individual of the reproductive caste, called a "supplementary reproductive"; *j*, wingless, apterous, or second-form individual of the reproductive class, also known as a supplementary reproductive. (After Kofoed *et al.*)

the termites in special compartments of the termitarium. The spores of the fungus are ingested by the termites, are passed uninjured through the intestinal tracts of the workers, and thus are introduced automatically into the substrate. The fungus is used almost exclusively as food for the young and for the royal



FIG. 31.—A termitarium of the Philippine mound-building termite, *Macrotermes gilvus* cut open to show the spongelike fungus gardens. (After Kofoid et al.)

castes. The soldiers and workers do not feed upon the fungus but use other plant material.

Very little is known about the identity of the fungi cultivated by the termites. Fruiting bodies of *Xylaria* and *Volvaria* have been found associated with the gardens and are thought by some to be identical with the fungi in the gardens. However, very few of the fungi associated with the termites have been

studied extensively, and more careful taxonomic studies are desirable.

Many species of termites that do not cultivate fungi in special gardens feed on wood decayed by fungi. Others are able to destroy healthy wood, but it has been observed that the wood adjacent to the tunnels made by the termites is always infested with fungi. This suggested the possibility that even these species might be more closely associated with fungi than previously supposed. Accordingly, Hendee (1933) has made a study of the association of fungi with three species of termites [*Kalotermes minor* (Hagen), *Reticulitermes hesperus* Banks, and *Zootermopsis angusticollis* Hagen] that are destructive to buildings. Hendee isolated 33 known genera and 20 nonsporulating cultures of fungi from the termites or their burrows. *Penicillium* and *Trichoderma* were the genera most constantly present, but there was no evidence of a specificity of association as the same species are found in nature independent of termites. The termites, however, are never found without the fungi. There was no correlation between the fungi present and the species of wood attacked by the termites. It was shown that the termites regularly transported viable spores into locations to which they would not have access otherwise and that the fungi invaded the wood a short distance ahead of the excavations of the termites. The fungi associated with some termites (*Reticulitermes* and *Zootermopsis*) causes a definite wood decay.

In a further series of nutritional experiments with *Z. angusticollis*, Hendee (1935) has shown that the fungi play an essential role in the natural diet of the termites. This was done by feeding the termites on various fungus-containing and fungus-free diets and measuring the effects of each by several different criteria. In interpreting the role played by fungi, Hendee states:

The fungi offer a source of proteins. They probably supply vitamins which are essential to the normal growth and development of termites. Through the secretion of extracellular enzymes they may render the wood itself more available. It is not known what effect the fungi may have on harmful extractives of the wood.

Termites and Protozoa.—A discussion of the symbiotic life of termites would not be complete without a brief consideration

of the symbiotic protozoa and the part they play in the nutrition of these insects. It is now known that practically all species of wood-eating termites harbor in their intestinal tracts large numbers of protozoa. The significance of these symbiotic protozoa has been studied extensively by Cleveland (1923 to 1934), who proved experimentally that they are necessary for the digestion of the cellulose of the wood on which the termites feed. Cleveland (1924, 1925*b*, 1925*c*) found that the termites could be freed of protozoa by subjecting them to high temperatures (24 hours at 36 degrees centigrade) or by oxygenation. Such defaunated termites died of starvation within 10 to 20 days if kept free of protozoa, but if recontaminated with the protozoa of their normal intestinal fauna, they continued to live and develop normally. Mansour (1934*b*) has questioned Cleveland's conclusions and adduces evidence to support the view that the relationship is not mutualistic symbiosis but one of commensalism, in which both termites and protozoa get all their required food from the wood and derive no benefit from the association. However, the more recent work of Hungate (1936, 1938) supports the conclusion that the protozoa are essential in the digestion of cellulose. This author has made extensive quantitative studies of wood digestion in faunated and defaunated termites (*Zootermopsis nevadensis* and *Z. angusticollis*). He concludes the protozoa are responsible for digesting approximately two-thirds of the total amount of wood that is utilized and that the remaining one-third is available without the aid of the protozoa. The latter quantity, however, is not sufficient to meet the energy requirements of the termites, which is shown to be seven-eighths of the total amounts of wood digested by all agencies.

Cleveland (1925*a*, 1928) cultivated reproductive colonies of termites for more than 18 months on a diet of filter paper and concluded that they could live indefinitely on a diet of pure cellulose, although he was at a loss to account for the source of nitrogen required for their fortyfold increase in weight. He concluded that "they must be able in some way to fix atmospheric nitrogen which they use in manufacturing proteins; or else, contrary to the current opinion, they must be able to transform carbohydrates into proteins." He was unable to demonstrate fixation of atmospheric nitrogen; and as the other alternative is obviously impossible, the question of the source of nitrogen was

left unanswered. Cook and Scott (1933) have questioned the ability of termites to survive on a diet that includes carbohydrates, proteins, salts, and vitamins A, B, D, and G. These authors used as criteria the viability and the group weight of nonreproductive colonies over a relatively short period of time and placed considerable weight on the presence or absence of cannibalism. These criteria are subject to some criticism because, as pointed out by Hendee (1935), cannibalism often occurs in normal colonies on a natural diet of rotten wood. This might account for a decrease in weight and numbers during the first few months that could not be attributed to a deficient diet. Even though the termites may not be able to survive indefinitely on a diet of pure cellulose, as claimed by Cleveland, his data indicate that the colony lived and thrived for a surprisingly long time on a diet of pure cellulose.

Uvarov (1928), in discussing the nutrition of termites, commented: "The most difficult problem is not the digestion of cellulose, but how to discover the means by which the deficient nitrogen is obtained. The only suggestion made in this direction is that symbiotic microorganisms are able to fix atmospheric nitrogen, but there is no evidence of any kind in support of this theoretically very reasonable view."

A possible explanation of the apparent ability of termites to thrive on a nitrogen-deficient diet has been offered by Leach and Granovsky (1938). The hypothesis is based on a possible nitrogen cycle within the termite colony. The symbiotic microorganisms, including protozoa, bacteria, and spirochetes, are localized in an enlargement of the hind-gut *posterior* to the point where the Malpighian tubules enter the intestinal tract (Fig. 32). The urates and other nitrogenous waste products excreted by the Malpighian tubules are probably utilized as food by the microorganisms and elaborated into protoplasm. Since proctodaeal, or anal, feeding (the feeding on excreta of other individuals) is common among termites, the bacteria and protozoa or their dead bodies obviously are consumed by some individuals of the colony. Refaunation has been considered the principal function of anal feeding. It may also play a part in the nitrogen economy within the termite colony. The synthesis of protoplasm from urates by the protozoa and the utilization of the dead protozoa as food by the termites would permit the same nitrogen supply

to be used over and over. In this way, the metabolic processes of the termite colony could continue indefinitely as long as carbohydrates were available. The maximum size of the colony would, of course, be limited by the amount of nitrogen originally available in the bodies of the founders of the colony. Within this limit, such a nitrogen cycle within the termite colony would permit the colony to live for a very long time on a diet apparently consisting of cellulose only.

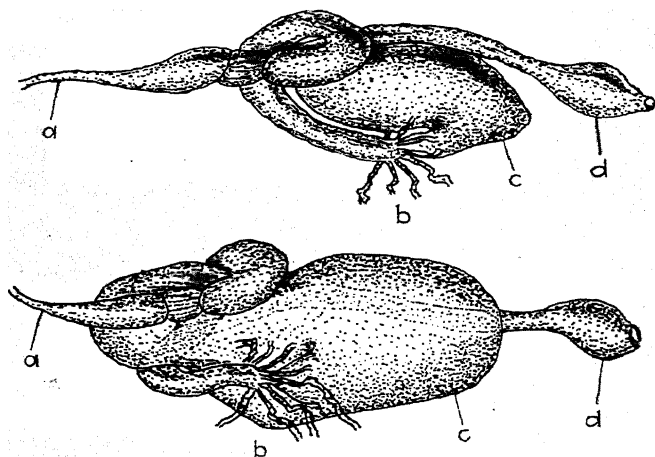


FIG. 32.—Intestinal tracts of worker and soldier castes of *Calotermes flavicollis* Fabr. a, oesophagus; b, Malpighian tubules; c, enlarged portion of hind-gut in which protozoa are localized; d, rectum. Note that the enlargement containing the protozoa is behind the point of entrance of the Malpighian tubules. (Redrawn from Buchner's "Tier und Pflanze in Symbiose," by permission of Gebrüder Borntraeger.)

It is also probable that some nitrogenous matter derived from dead protozoa within the digestive canal is absorbed directly through the anterior region of the hind-gut. Child (1934), in a study of the internal anatomy of termites, concluded that some portion of the voluminous organ formed by the vestibule, large intestine, and caecum must absorb the digested food material. This organ with its large expanse of thin walls is bathed by the hemolymph and is excellently suited to the purpose. The conclusion is supported also by the fact that the

wood particles are not completely digested until they reach the hind-gut. If nitrogenous compounds derived from the dead protozoa are absorbed in this region, the nitrogen is probably used over and over by a single individual, thus increasing greatly the efficiency of the insect's nitrogen economy.

Termites appear to have developed a highly effective method of conserving nitrogen. In all probability, the fungus-cultivating

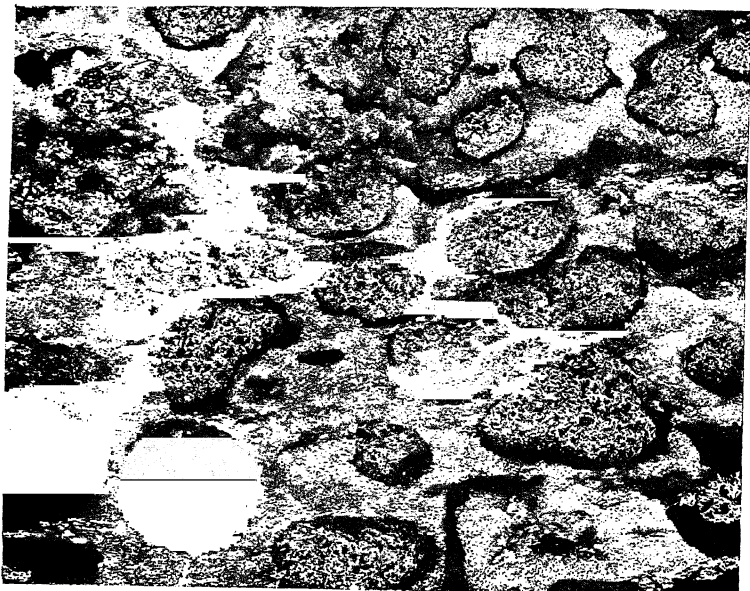


FIG. 33.—A sectional view of a portion of a nest of the fungus-cultivating ant *Atta vollenweideri* showing the chambers containing the spongy fungus gardens. (After Wheeler, with permission of Harcourt, Brace & Company.)

habit of termites is also based on a similar nitrogen economy. It is well known that the fungi cultivated by the termites grow almost exclusively on the excreta. The fungi transform nitrogenous waste products into fungus protoplasm, which is in turn consumed by the termites to form more excreta on which to grow more fungi.

Ants and Fungi.—Ants are among the most highly developed insects, and, according to Wheeler (1923), "the number of individual ants is probably greater than all other insects."

They are highly social in their life habits. The leaf-cutting ants and the fungi that they cultivate constitute one of the best examples of ectosymbiosis of insects and fungi. Other species of ants are often associated to a lesser extent with fungi, and a few species have been suspected of being vectors for plant pathogenic fungi. Wheeler (1907, 1923, 1937) and Weber (1937, 1938) have given such a full account of the biology of these ants and their associated fungi that only the briefest outline will be given here.

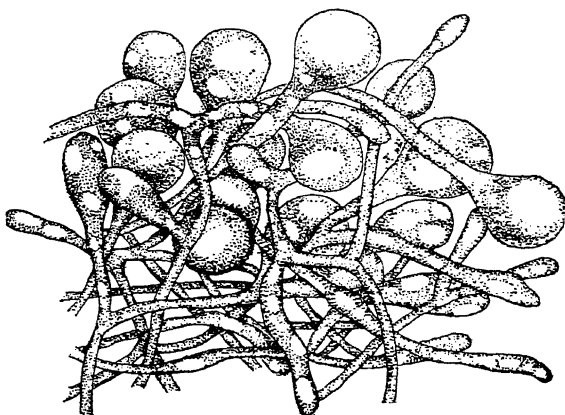


FIG. 34.—“Bromatia” or globular swellings of the hyphal tips of the fungus cultivated by ants. The ants feed upon the bromatia in preference to the normal mycelium. (From Wheeler after Carlos Bruch, with the permission of Harcourt, Brace & Company.)

The fungus-cultivating ants consist of about a hundred tropical or subtropical, exclusively American species belonging to the tribe Attini. These ants live in enormous colonies in extensive underground nests. They forage on all kinds of vegetation for long distances in every direction. Leaves and other succulent plant parts are cut and transported into underground nests where they are used as a substrate for the cultivation of the associated fungus. The plant fragments are worked into a spongy pulp on which a luxuriant fungus growth is maintained (Fig. 33). A different fungus appears to be cultivated by each species or group of closely related species of ants.

As the fungi grow in the "gardens," no spores are formed, but characteristic club-shaped hyphal tips composing the so-called "bromatia" are produced in abundance. The bromatia constitute the edible portion of the fungus, and, as the swollen ends are eaten, new ones are constantly formed (Fig. 34).

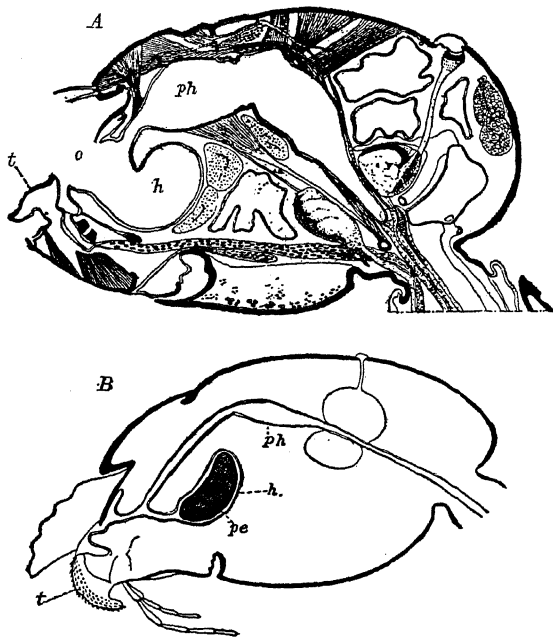


FIG. 35.—Sagittal sections through the ant head showing the infrabuccal pouch and pellet. A, a queen of *Lasius niger* with the mouth open; B, a queen of *Comptonotus brutus* with the mouth closed; t, tongue; o, oral orifice; ph, pharynx; h, infrabuccal pouch; pe, infrabuccal pellet. (After Wheeler, with permission of Harcourt, Brace & Company.)

The identity of the cultivated fungi has not been determined definitely, but the fruiting bodies of certain Agarics and of Xylaria have been found associated with the fungus gardens. Möller in 1893 described an Agaric found associated with the nests of *Acromyrex disciger* Mayr. and named it *Rozites gongylophora*. Spegazzini in 1899 found a stroma growing from the nest of *Acromyrex lundii* and named it *Xylaria micrura*. Bruch (1922), also, has reported this fungus associated with the same

species of ant. Wheeler (1907) described a yeastlike fungus cultivated by *Cyphomyrmex comalensis*, named it *Tyridiomyces formicarum*, and placed it among the Exoascaceae, although no ascospores were observed. These papers and others dealing with the identity of the ant-cultivated fungi are reviewed by Weber (1938) who has also described *Lentinus atticulus* Weber as the fungus cultivated by *Atta cephalotes* L.

The technique of cultivation of the fungi by the ants has been described in detail by Wheeler (1923). When the ants swarm and new colonies are established, the female retains a mass of the fungus and leaf tissue, or "spawn," in her infrabuccal pouch (Fig. 35). After mating and burrowing into the soil, this spawn is used in starting a new fungus garden. Fresh leaf tissue is added to the spawn, and the new garden is fertilized with excrement and often with eggs which are crushed and mixed with the substrate (Fig. 36). The garden is later cared for by the workers, and the larvae feed exclusively on the bromatia.

There is a striking similarity between the fungus-cultivating habits of the ants and of the termites. Both cultivate their fungi in special fungus gardens on an organic substrate. The termites utilize their excrement as a substrate for the fungi while the ants prepare a special substrate of plant tissue. The fungus gardens of the ants growing on the substrate, however, are constantly fertilized by excreta, and Weber considers "that the role of the substratum is largely passive, affording mainly a convenient framework for the nutritive fecal droplets and the mycelium which grows on them. . . . A cycle is thus set up which is theoretically endless, the ants feeding on a fungus which in turn grows on the excrement of the ants."

The infrabuccal pouch, an organ characteristic of ants, is an interesting structure and demands further consideration from the standpoint of fungus associations. This pouch (Fig. 35) is used by ants in general as a screen and repository for particles of solid material not suitable for ingestion. When the pouch has become filled, the contents are discharged in the form of pellets which often contain, among other things, large quantities of fungus spores as shown by Bailey (1920) (Fig. 37). He worked with specimens preserved in alcohol and did not determine the viability of the spores taken from fresh pellets. Germination experiments reported by Leach and Dosdall (1938), however,



FIG. 36.—Photographs showing a queen ant (*Moellerius heyeri*). A, in the act of laying an egg, B, placing an egg in the fungus garden; C, in the act of fertilizing the fungus garden by defecation. (After Wheeler, with permission of Harcourt, Brace & Company.)

show that some of the spores in the infrabuccal pellets of *Formica fusca* var. *subserica* Say are viable. Because ants are constant visitors to all parts of plants, both diseased and healthy, these observations are of considerable significance. Ants found constantly associated with a fungus plant disease should be viewed with suspicion. Although ants have not been incriminated as vectors of plant pathogens in many instances, there are a few cases on record. For example, Dade (1927) has reported that ants are important vectors of the mealy-pod disease of cocoa

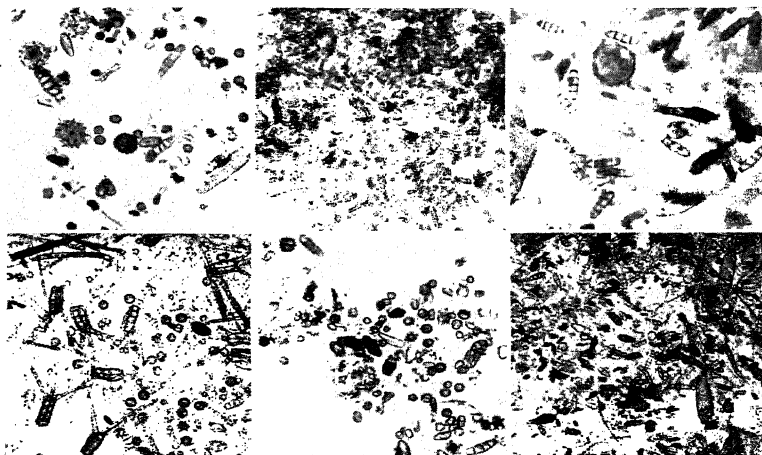


FIG. 37.—Photomicrographs showing the fungus spores found in the infrabuccal pouches of six different ants. The pellets were dissected out, crushed in a drop of water on a slide, and photographed. (After Bailey.)

in the African Gold Coast. This disease is caused by a Phycomycetous fungus (*Trachysphaera fructigena*), which forms masses of spores on the surface of affected pods. Ants regularly feed upon the diseased pods, and they were observed transporting the spores. The ants were considered effective agents of spore dissemination, although it was recognized that the spores may also be scattered by wind and rain.

There is still another way in which ants may be involved indirectly in the transmission of plant diseases. It is well known that many species of ants utilize as food the so-called "honeydew" of aphids and other homopterous insects. The

honeydew is in reality the excrement of these insects and is sought by the ants because of its high sugar content. The plant juices ingested by the aphids and scale insects have a high water and sugar content in relation to proteins. In order to obtain the required amount of protein food, an excess of water and sugar must be ingested. This excess is voided in the form of the sugary solution known as honeydew. Many species of ants have acquired the habit of caring for aphids, mealy bugs, and other insects so that they may utilize the honeydew to the best advantage. Individual aphids, for example, are transported by ants from plant to plant and placed in the proper location for the establishment of colonies. Some ants even collect the aphid eggs in the fall and preserve them in their nests over winter. So highly specialized is this relationship that Linnaeus referred to aphids as the "dairy cattle of the ants." As aphids are the most common vectors of virus diseases, it is evident that the association is one that should not be overlooked in the study of insect transmission of virus diseases and of the control of the insect vectors.

Ants that attend the pineapple mealy bug [*Pseudococcus brevipes* (Ckl.)], a toxicogenic insect causing a destructive wilt of pineapple, are essential for their normal development, according to the work of Carter (1933a). No successful attempts to control aphids or mealy bugs by controlling the ants have been reported. Unfortunately, effective control of the ants often is more difficult than direct control of the aphids or mealy bugs.

Septobasidium and Scale Insects.—Couch (1931, 1939) has described in considerable detail a unique case of mutualistic symbiosis between the fungus, *Septobasidium burtii* Lloyd, and a scale insect, *Aspidiotus osborni* New. and Ckl. *S. Burtii* is a well-known Basidiomycete occurring on the branches of many kinds of trees (Fig. 38). Although the injury to the tree is slight, the fungus has been considered by some to be a parasite. Others have held it to be an epiphyte, and still others have considered it a saprophyte living on the excretions of the scale insects. Couch has demonstrated that the fungus and the scale insects live symbiotically at the expense of the host plant. The scale insects live among the folds of the fungus where they are protected against enemies and unfavorable environment. In this home furnished by the fungus, the scale insects suck the

juices from the host plant, grow, and multiply. In return, the insects supply nourishment for the fungus. The mycelium of the fungus penetrates the body cavities of a number of the insects and forms numerous coillike haustoria. The nourishment absorbed from the insects through these haustoria supports the growth of the fungi. The fungus is disseminated entirely by young insects that become infected with the fungus and migrate

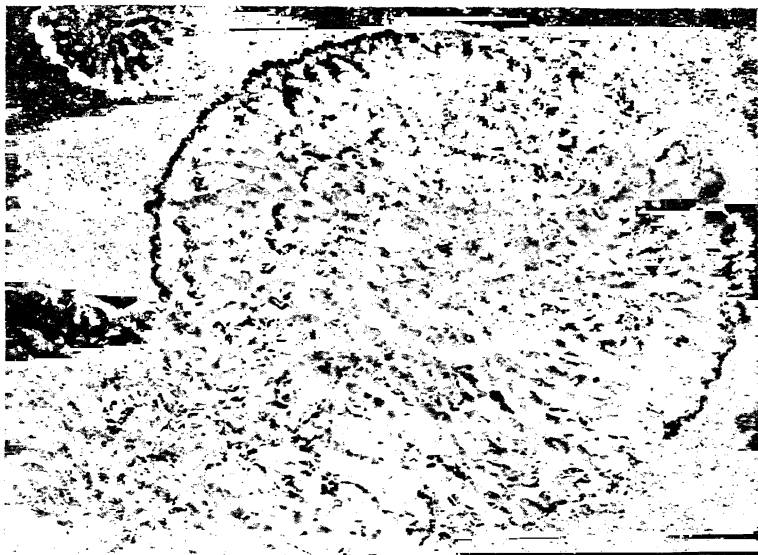


FIG. 38.—*Septobasidium Burtii* growing on the bark of *Quercus palustris*. Surface view showing the radiating ridges and depressions that mark the openings to tunnels occupied by the scale insects. Approx. 4X. (After Couch, by permission of University of North Carolina Press.)

to new localities. Unless the insects are infected when young, they remain free of the fungus, for the older ones are immune to infection. Some individual insects die and are absorbed by the fungus; others apparently digest the fungus haustoria, and others are free from infection. In Fig. 39 is reproduced Couch's diagrammatic representation of this symbiotic relationship. Similar symbiotic structures, varying with the species of fungus and insect, have been reported by Couch.

This symbiotic association appears to be universal in the genus *Septobasidium*. Couch (1935, 1939) has demonstrated it for



FIG. 39.—A diagrammatic representation of the symbiotic association of the fungus *Septobasidium burtii* and the scale insect *Aspidiotus osborni*. The insect derives protection from the fungus, and the fungus derives its food by parasitizing some of the insects. (After Couch, by permission of the University of North Carolina Press.)

at least 33 different species. Some species of *Septobasidium* are associated with only one species of scale insect while others may be associated with several species. In general, those species

of *Septobasidium* occurring on a wide variety of host plants are more likely to be associated with several species of scale insects.

Bacteria and Dipterous Insects.—That type of endosymbiosis in which the microsymbiote lives in the lumen of the intestinal tracts or in intestinal diverticula is well illustrated by the symbiotic relations between dipterous insects and bacteria, many of which are involved in destructive plant diseases. Several of these insects and their symbiotic relationships, including the seed-corn maggot, the apple maggot, and the olive fly, are described in detail in Chap. VI.

The Drugstore Beetle and Symbiotic Yeast.—This is the best-known example of endosymbiosis in which the microsymbiote has become intracellular and is found in mycetocytes in the intestinal wall or in the walls of intestinal caeca. The relationship between the drugstore beetle (*Sitodrepa panicea* L.) and its symbiotic yeast has been studied extensively by Buchner (1921). This beetle feeds on a variety of food materials, especially dried plant and animal products. Digestion of the food materials is accomplished through the aid of a symbiotic yeast (*Saccharomyces anobii* Buchner) that is closely and constantly associated with the intestinal tract, being found in the lumen and also in the epithelial cells of the larval intestine as well as in the cells of special caeca in the imago. The yeast is transmitted from generation to generation through surface-contaminated eggs. The microsymbiotes are found in great numbers in two intersegmental pouches; from there, they are discharged into two vaginal pockets from which they reach the eggs in the process of oviposition (Fig. 40). The eggs, as they are laid, press against the pockets and become surface-contaminated with the yeast. When the eggs hatch, the larvae become contaminated from the yeast cells adhering to the surface of the egg shells.

Intracellular Symbiosis in the Homoptera.—Intracellular symbiosis reaches its highest development in the Homoptera where the condition is practically universal. The microsymbiotes are found deep in the body tissues either free in the hemolymph or in individual mycetocytes or in mycetomes. Symbiosis of this type has been rather extensively investigated by Pierantoni, Sulc, and Buchner. Brain in 1923 summarized the literature to that date and drew up a provisional classification

of the described microsymbiotes, establishing a number of new genera. The generic names for these microsymbiotes living free in the hemolymph or connective fat tissue were formed by combining the suffix "cola" with some other descriptive word, usually the genus or class name of the insect. For those microsymbiotes constantly inhabiting definite mycetocytes or myce-

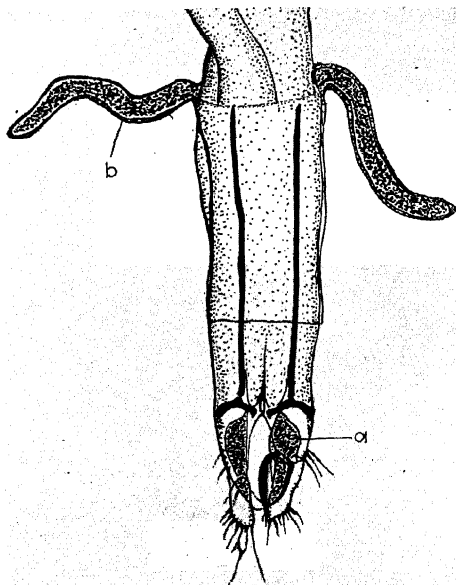


FIG. 40.—The ovipositor of *Sitotrepa panicea* showing the vaginal pouches (a) filled with symbiotic yeast cells that contaminate the eggs at time of oviposition, and the long intersegmental pouches (b) in which a constant supply of the yeast cells are maintained. (After Breitsprecher, from Buchner's "Tier und Pflanzen in Symbiose" with permission of Gebrüder Borntraeger.)

tomes, the suffix "myces" is used. Thus, *Lecaniocola*, *Cicadocola*, and *Cicadomyces*, *Aphidomyces*, etc. Forty-two species of microsymbiotes belonging to 13 genera were described.

A typical representative of this type of symbiosis has been described in some detail by Granovsky (1929) who studied the intracellular symbiotes of *Saissetia oleae* (Bernard) taken from oleander plants growing in a greenhouse. The yeastlike symbiote (Fig. 41a) was found constantly associated with all stages in the

development of the insect, although they were more abundant in half-grown nymphs and in young females. No similar organism was found on the leaf surface, in the honeydew secreted by the insect, or elsewhere outside the insect body. The microsymbiote was not grown in artificial culture, but it was seen to multiply by budding during the first 24 hours when suspended in a hanging drop of normal salt solution.

Histological studies showed that the microsymbiotes occurred free in the hemolymph as well as in the cells of the connective fat tissue. As the amount of fatty tissue decreases, in mature females, the microsymbiotes decrease in abundance, probably by death and disintegration. The association of the micro-

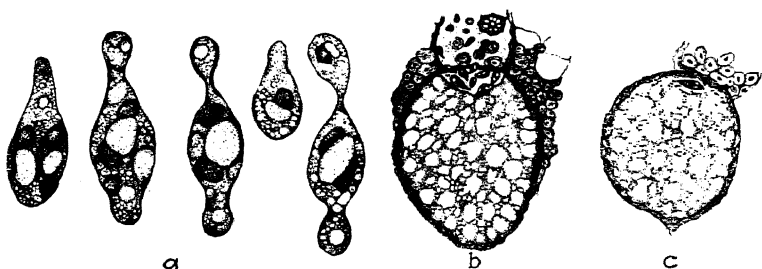


FIG. 41.—*Lecaniocola saissetiae* the yeastlike symbiote of *Saissetia oleae*. *a*, the symbiote showing multiplication by budding in a normal salt solution within 24 hours after removal from the insect; *b* and *c*, the symbiotes invading the developing egg from the nurse cells. (After Granovsky.)

symbiotes with the fatty tissues has suggested that they may aid in the digestion of the reserve foods during the reproductive life of the insect. Congenital transmission is accomplished by internal infection of the eggs. This apparently occurs through the anterior end of the egg through the nurse cells (Fig. 41*b*) or by direct penetration near the anterior pole. The organism was described and, following Brain's system of nomenclature, named *Lecaniocola saissetiae*.

Perhaps the most highly specialized type of symbiosis of this kind is represented by that found in the Aphididae. All members of this family are equipped with a large mycetome divided into two longitudinal halves. This structure was first observed and described by Leydig in 1850 and was further studied by Huxley (1859), who applied the name "pseudovitellus" to it. Pierantoni

(1909, 1910) and Sulc (1910) were the first to demonstrate the presence of symbiotic microorganisms in the structure, Sulc introducing the terms "mycetome" and "mycetocyte." The most recent and most exhaustive study of the mycetome of the Aphididae is that of Uichanco (1924), who has described in detail the method of congenital transference of the mycetome and the included symbiotes. The follicular epithelium becomes infected by the symbiotes during the early stages of development and harbors them in a dormant condition. During the earlier cleavage stages of the egg, the dormant symbiotes come in contact with the egg yolk and are stimulated to rapid multiplication. As a result of the growth of the symbiotes, there is a swelling of a portion of the follicular epithelium; the symbiotes break through the thin epithelial cells, invade the posterior portion of the egg cavity, and appropriate the yolk mass enclosed there. In this way, every individual receives its complement of mycetocytes and the associated microsymbiote.

After birth of the aphid, the mycetocytes cease to multiply but increase to approximately double their original size. The symbiotes, however, multiply extensively. As the insect reaches maturity, the mycetocytes begin to degenerate until the death of the insect, when only a few are left.

The mycetome is present in every individual aphid, both male and female, regardless of method of reproduction. This constant association of the microsymbiotes would indicate that they play an important role in the biology of the aphids, but their function is still an unanswered question.

Rickettsia.—This name is applied to a group of very small bacteriallike microorganisms that are found living symbiotically in the alimentary canal of many insects and other arthropods. They are found frequently within the epithelial cells where they apparently cause no injury although some workers consider them parasitic instead of symbiotic. They also may invade other tissues, including the salivary glands from which they enter the salivary secretions. Moreover, most of them appear to be congenitally transmitted to successive generations through the eggs. Rickettsia are Gram-negative and stain poorly with aniline dyes but take the Giemsa stain readily. They are usually less than half a micron in diameter. It is extremely difficult if not impossible to cultivate them in artificial media.

A number of *Rickettsia* are pathogenic to animals, causing such diseases as typhus fever, the Rocky Mountain spotted fever, and trench fever. The generic name *Rickettsia* was first used to designate the pathogen of typhus fever (*Rickettsia prowazekii*). This binomial honors Ricketts and Prowazek, two investigators who died of typhus fever while investigating the disease. The pathogenic *Rickettsia* are all transmitted by arthropods and are adapted to alternate existence in arthropods and in higher animals.

Swezy and Severin (1930) have reported *Rickettsia*-like microorganisms in the epithelial cells and lumen of the intestines of the sugar-beet leaf hopper (*Eutettix tenellus*). These workers suggested a possible relationship of the *Rickettsia*-like organisms to the cause of curly top of sugar beets. There is, however, no convincing evidence that the microorganisms are concerned in causing the disease. No plant parasitic *Rickettsia* are known.

In the preceding discussion, it has been possible to touch only briefly upon a few of the most important aspects of the subject of symbiosis between insects and microorganisms. The subject is extremely complicated, and although a large body of information has accumulated in recent years, the principles underlying the phenomenon are only imperfectly known. Relatively little accurate information is available on the physiological bases of symbiosis, and the biological significance of the associations is not well understood. It is evident, however, that symbiosis between insects and microorganisms is a relatively common phenomenon and that such associations are often directly concerned in the spread and development of plant diseases. For these reasons, it is obvious that the subject should not be neglected by anyone interested in insects as vectors of plant pathogens.

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CHAPTER IV

THE RELATION OF INSECTS TO THE SPREAD AND DEVELOPMENT OF PLANT DISEASES

HISTORICAL

The parasitic nature of the most common plant diseases was not recognized until the middle of the nineteenth century. Prior to this time, the autogenetic theory of the cause of plant diseases prevailed. Fungi were known to be associated with the diseased plants, but they were thought to be a result of the disease rather than the cause. For example, such parasitic fungi as the rusts and smuts were visualized as products of the disordered sap of the diseased plants. The spores of these fungi had been seen through the microscope, but it was not known that they could germinate and infect healthy plants. It is only natural that few or no significant observations concerning the role of insects in the spread and development of plant diseases were made until the true nature of parasitic plant diseases was recognized.

The publications of De Bary, beginning in 1853, proved conclusively the parasitic habits of fungi associated with diseased plants. De Bary's work and that of his contemporaries, Julius Kühn and the Tulasne brothers, changed the whole concept of the nature and cause of plant diseases. During this period, Pasteur and Koch proved and established the germ theory of animal diseases, and in 1881 Burrill demonstrated, for the first time, that bacteria could cause plant diseases. These fundamental discoveries greatly stimulated the study of both plant and animal diseases, and rapid advances were made. At first the greatest emphasis was placed on the discovery and proof of the causes of the various diseases. Many new microorganisms were found and described, and their pathogenicity was proved. Later, attention was turned to the means of spread and to the factors influencing the development of diseases. It was then that pertinent observations and experiments on the role of

insects were made. This does not mean that the idea of insect transmission of diseases did not appear until the parasitic nature of diseases was recognized. Indeed, there are numerous early references to the subject and expressions of opinion that insects were concerned in the development of diseases of both plants and animals. Some of these theories were based on careful observation and bore some elements of truth. Many of them, however, were imaginary and inaccurate, and none of them received much credence at the time. It was only after later experimentation had established the real nature of the relationships that those earlier observations, which were so near the truth, appeared significant. A classic example is that of the Italian physician Mercuriales, who, in the sixteenth century, formulated a theory that the plague (Black Death) was spread by flies. He held that the disease was transmitted by flies feeding upon excretions of diseased individuals and later defecating upon human food. Although his theory was not based on experimental evidence and was incorrect, it is nevertheless based upon a concept of the infectious nature of diseases that was not fully appreciated until 250 years later.

The first experimental proof of insect transmission of a plant disease was reported in 1891 by Waite, who showed that bees and wasps, while visiting pear blossoms in search of nectar, were active vectors of the bacteria causing fire blight of pears. Seven years earlier, Forbes (1884) had observed the tarnished plant bug feeding on blighted pear twigs and had expressed the opinion that the insects transmitted the disease from tree to tree. No experimental proof was offered by Forbes, but the correctness of this theory was verified by Stewart (1913) nearly thirty years later.

The proof that the troublesome fire-blight disease was disseminated by bees and other insects served to focus the attention of plant pathologists on the problem. Although Waite's work was not extensive and he left many pertinent questions unanswered, he experimentally transmitted the disease by means of bees from one blossom cluster to another and protected other blossoms from infection by caging them and excluding the insects. He demonstrated the presence of the pathogen in and on the mouth parts of bees and wasps caught in the orchard. Even though the work was fragmentary and incomplete, it marks the

beginning of experimental investigation of insects in relation to plant diseases and has furnished the necessary stimulus for further study.

In 1901, Takami, working in Japan, showed that the dwarf disease of rice, now known to be a virus disease, was brought about as a result of the feeding of a leaf hopper (*Nephotettix apicalis* Motsch.). This was a discovery of fundamental significance; but partly because the work was published in the Japanese language, it was generally overlooked until other investigators working with similar diseases had demonstrated that virus diseases were generally transmitted by insects. In fact, according to Katsura (1936), Takami in 1901 did not have the concept of the leaf hopper acting as a vector of an infectious disease but rather considered the insect as the sole cause of the disease. Only after several years of subsequent work was the correct nature of the relationship realized and published in 1908 and 1909 in the reports of two Japanese experiment stations.

Ball, in 1906, five years after the publication of Takami's paper, demonstrated that curly top of sugar beets was associated with the leaf hopper [*Eutettix tenellus* (Baker)]. The subsequent work of Shaw (1910), Smith and Bonquet (1915), and others soon demonstrated the infectious nature of the disease and the true role of the leaf hopper as a specific vector of curly top. At about this time, Allard (1912) showed that a virus disease of tobacco was transmitted by aphids. These discoveries of Ball and Allard marked the beginning of a long series of investigations that has shown the great importance of insects as vectors of a large number of virus diseases. They opened up a new and fruitful field of research.

Similar developments were being made in the field of animal pathology at approximately the same time that plant pathologists were recognizing insects as important vectors of plant diseases. As early as 1878, Manson had shown that mosquitoes were the vectors of *Filaria bancrofti*, the nematode worm causing elephantiasis of man. This early discovery of the transmission of a disease of man by an insect had a profound influence on the trend of development in medical science. Manson, convinced of the potential significance of mosquitoes as vectors of disease, stimulated and encouraged Ross in his classic studies on the transmission of malaria by mosquitoes. The pioneer work of

Manson and the concepts based on it mark the real beginning of the scientific study of insect transmission of disease.

In 1893, Smith and Kilbourne published their classic work on the transmission of the Texas fever of cattle by the cattle tick. This very thorough investigation of the transmission of an animal disease by a small arthropod also established new concepts and stimulated further studies in the field of medical entomology.

Bruce, in 1895, demonstrated the association of the tsetse fly and the nagana disease of cattle in Africa; this was soon followed by the work of Ross (1898) and others proving that mosquitoes were responsible for the transmission of malaria. These epoch-making discoveries led to the proof in 1900 by the United States Army Yellow Fever Commission that yellow fever is transmitted by mosquitoes and is dependent upon these insects for its existence. (For a more complete summary of insect transmission of animal diseases, see Chap. XVI.)

Further advances in the field of plant pathology were made in 1909 and 1910 when Petri showed that the olive fly (*Dacus oleae*) could transmit the olive-knot disease. The intricate symbiotic association of this insect and several species of bacteria, including the olive-knot pathogen, illustrates a high degree of adaptation between insects and bacteria that is of great significance in plant pathology.

In 1911, E. F. Smith reported proof of the transmission of bacterial wilt of cucurbits by the striped cucumber beetles. This was followed by more complete reports by Rand (1915) and by Rand *et al.* (1916, 1920) who proved conclusively that the cucumber beetles were vectors of the disease and probably the only means of transmission of the disease in nature. They showed also that the pathogenic bacteria were preserved over winter in the bodies of the insects and that the overwintered insects were responsible for primary infection in the spring. The disease appears to be entirely dependent upon the insects for its spread and survival.

In the same year, Norton (1911) published a paper of considerable historical interest. This was a short article entitled "The health of plants as related to insects." It apparently was the first article of a general nature ever published on the subject of insects in relation to plant diseases. Norton reviewed

briefly the association of insects and plant diseases known at that time and called attention to the importance of the subject and the need for further study. He classified the action of insects on plants according to the following categories:

1. Direct injuries.
 - a. Physiological troubles following injury.
 - b. Diseases caused by the presence of insects or their products.
2. Diseases indirectly caused by insects (by dissemination of the pathogens).

Two years later, Heald (1913) published a well-organized summary of the agencies and methods of dissemination of plant pathogenic fungi. Using this article as a starting point, Gardner (1918) published a somewhat more extensive treatment of "The mode of dissemination of fungous and bacterial diseases of plants." In both these papers, the growing recognition of the importance of insects in the dissemination of plant pathogens is obvious.

A discovery of fundamental interest was made by Ball in 1918 when he demonstrated that much of the so-called "tipburn" of potatoes was caused by the feeding activities of the potato leaf hopper (*Empoasca fabae* Harris). His discussion of this injury gave rise to a new concept in plant pathology and led to further investigation of the toxic effects of insect feeding punctures and the many obscure diseases that are associated with such injuries. Since then, numerous diseases of similar nature have been described, and much has been learned about these "toxicogenic" insects and the diseases that are caused by them.

In the 30 years following the pioneer work of Waite, a considerable amount of work on the subject of insect transmission of plant diseases appeared in the literature. These reports were widely scattered in periodicals and bulletins. In 1920, Rand and Pierce published an article entitled "A coordination of our knowledge of insect transmission in plant and animal diseases." In this 42-page article, "written primarily in the interests of plant pathologists," the authors summarized and coordinated the literature in both fields of work. They recognized and pointed out certain principles common to insect transmission of both animal and plant diseases and used these principles as a basis for classification of the known phenomena of insect transmission. As a direct result of the interest created

by this paper, the American Phytopathological Society and the American Association of Economic Entomologists at their annual meetings in Toronto, Canada, on Dec. 31, 1921, held a joint symposium on "Insects as disseminators of plant diseases" (Rand, Ball, Caesar, and Gardner 1922). This symposium created still greater interest in the subject and made evident the need for closer cooperation between plant pathologist and entomologist.

In 1921, there appeared a book entitled "Tier und Pflanze in Intracellular Symbiose," written by Buchner, a German entomologist who for a number of years had been investigating the phenomena of symbiosis between insects and microorganisms. In 1930, the book was revised and its scope expanded under the title "Tier und Pflanze in Symbiose." These books bring together for the first time the scattered information on the fundamental phenomena of symbiosis between insects and microorganisms; although the subject is not treated from the viewpoint of plant pathology, the problems dealt with are very significant in the study of insects in relation to plant diseases.

The increasing interest in the subject of insects in relation to plant disease was shown in 1929 when Böning published in Germany an extensive critical review of the literature on "Insekten als Ueberträger von Pflanzenkrankheiten." The growing importance of virus diseases from this standpoint is manifested by the preponderance of citations of literature in this field. In 1931 and 1933, Smith published summaries of the accumulated knowledge of virus diseases in which he devoted considerable space to the subject of insect transmission of viruses. These were followed by a general textbook of virus diseases of plants (Smith 1937).

The appearance of the destructive and spectacular Dutch elm disease in Europe and America and the proof of its dependence upon insects have, in a very dramatic way, focused the attentions of both entomologists and plant pathologist on the importance of insects as vectors of plant diseases. Investigations of this disease have demonstrated to a striking degree the necessity of cooperative effort in the study of insect-transmitted diseases.

The general subject of insects in relation to plant diseases was reviewed in 1935 by Leach, who pointed out its importance and promise as a field of research and urged its further develop-

ment. A growing interest in the subject was manifested in the symposium on insects in relation to plant diseases conducted jointly by the Entomological Society of America, the American Association of Economic Entomologists, and the American Phytopathological Society at Indianapolis, Ind., in 1937. In this symposium, various aspects of the subjects were treated by Granovsky (1938), De Long (1938), Kunkel (1938), Searls (1938), Leach (1938), Poos (1938), Ingram (1938), and Stevens (1938).

In general, the progress of plant pathology has paralleled that of human and animal pathology. The importance of insects in human pathology has led to the development of the extensive and rapidly growing field of medical entomology. The trend of events seems to indicate that a similar development is taking place in the science of plant pathology.

HOW INSECTS AID IN THE SPREAD AND DEVELOPMENT OF PLANT DISEASES

Insects influence the development of plant diseases in many different ways. For convenience the various types of association may be classified according to the particular function performed by the insect. Insects may be responsible for, or aid in:

1. Direct production of a disease without the help of a pathogen, usually by the injection of a toxic substance into the tissue of the plant.
2. Dissemination of the pathogen.
3. Inoculation of the susceptible with the pathogen.
4. Ingression of the pathogen into the susceptible.
5. Invasion of the susceptible by the pathogen.
6. Preservation of the pathogen.

Direct Disease Production.—Some insects, in feeding upon plants, cause only mechanical destruction of the tissues upon which they feed. Others cause injury to tissues some distance removed from the point of feeding. The degree of injury may vary from relatively local lesions to a general systemic effect on all parts of the plant. The exact mechanism of the injury is obscure in most cases, and there is need for much more study of this group of diseases. In some cases, there is convincing evidence that a toxic substance is injected into the tissues of the plant. In others, the physiological processes may be disturbed by local mechanical injury to vital tissues. As examples of nonparasitic diseases caused by the feeding of insects may be

mentioned hopperburn of potatoes, mealy-bug wilt of pineapples, the green-spotting diseases of pineapples, and psyllid yellows of potatoes. These and other similar diseases are discussed in more detail in Chap. V.

Dissemination of the Pathogen.—Most plant pathogens, under favorable conditions, produce relatively large quantities of inoculum. But before reinfection can take place, the inoculum must be dispersed to new plants or new infection courts. The spread of plant diseases and the development of epiphytotics depend to a great extent upon the efficiency with which the pathogen is disseminated. Inoculum may be disseminated in many different ways, wind, water, insects, man, and other animals being some of the most important agents. The relative importance of the different agents of dissemination varies greatly with different diseases. It is obvious that an accurate evaluation of the relative importance of the different methods is essential to an intelligent control program.

Wind is perhaps the most effective agent of dissemination of many kinds of inoculum, but the inoculum of many plant pathogens is not well adapted for wind dispersal. Spores of pathogenic fungi and cells of bacteria are often produced in sticky exudates that become hard on drying, precluding almost entirely the possibility of effective wind dispersal. In such cases, insects are known to play an important role.

The dissemination of pathogens is the most obvious way in which insects are concerned in the development of plant diseases. For this reason, although other functions have been recognized, the idea of dissemination has almost exclusively predominated all published literature of a general nature on the subject. The dissemination of the pathogen, however, is only one of several critical stages in the development of a disease.

Inoculation.—To “disseminate” is to scatter, broadcast, or diffuse. To “inoculate,” as the term is used in plant pathology, is to transport inoculum to a particular part of the plant where infection may result. Wind is a common agent of dissemination, but wind inoculation occurs only when, by chance, the inoculum falls on the right place. Most of the inoculum disseminated by wind never reaches the proper infection court and is consequently wasted. Many insects, because of their habits of visiting certain species of plants and specific organs of those plants, are not only

agents of dissemination but are also very effective agents of inoculation. They transport the inoculum to the most favorable place for infection with little loss of material. The honeybee and blossom blight of fruits, the sphinx moth and anther smut of pinks, the pollen-eating flies and ergot of cereals, and stigmatomycosis of various plants are well-known examples of this type of insect inoculation. More recently described associations of this nature are those involved in the so-called "fruit spoilage" of figs (Smith and Hansen 1931). Endosepsis (Caldis 1927), souring (Caldis 1930), and smut (Hansen and Davey 1932) all depend upon certain fig insects for introduction of the respective pathogens into the interior of the young fig fruit where infection occurs. These associations are treated more fully in Chap. VII.

The systemic disease of red clover caused by *Botrytis anthophila* Bond., described by Bondarzew (1914) and Silow (1933), is especially illustrative. The spores of this pathogen are transported by the bees that pollinate red clover. The spores, along with pollen grains, are deposited by the bees on the stigmas of the clover flowers, where they germinate. The mycelium of the pathogen, along with the pollen tube, grows and penetrates the ovary. The fungus continues to grow slowly and persists under the seed coat but does not destroy the seed. Infected seeds, when planted, give rise to systematically infected plants. The fungus grows out into the young flowers, many of which are blighted, and the spores are formed over the surface of the anthers (see Fig. 125). Bees visiting the blighted blossoms become contaminated with spores and spread the infection to other plants, completing a cycle closely paralleling the process of insect pollination of flowers.

Ingression.—All our higher plants have an effective natural protection against pathogens in the form of cuticle or cork. The entrance or penetration through this external covering by the pathogen is necessary for infection. This process of gaining entrance has been termed "ingress" by Whetzel (1929). Some pathogens are able to penetrate this protective layer by their own resources, but many depend upon wounds. The feeding or oviposition wounds of insects constitute one of the most important avenues of ingression for plant pathogens. The significance of insects as agents of dissemination and inoculation is greatly

enhanced when they serve also as agents of ingression. In some cases, this aid to ingression overshadows the importance of dissemination and inoculation. The role of the woolly aphid in the development of perennial canker of apple trees as described by Childs (1929) and McLarty (1933) is an excellent example of this type of relationship. These investigators have shown that *Gleosporium perennans* Zeller is strictly a wound parasite and that reinfection of a canker must take place each year. Although plenty of inoculum is present in the canker, reinfection does not occur in the absence of the aphid. This insect lives in the crevices of the canker and makes the wounds that are necessary for infection on each succeeding year's growth of callus. Thus the so-called "perennial" canker is actually annual, and the apparently perennial character depends entirely on reinfection each year through wounds made by the aphids which, although essential for the development of the disease, are of relatively little importance in dissemination of the spores.

Another example is afforded by the relation of soil insects to crown-gall infection. Banfield (1931) and Ricker and Hildebrand (1934) have shown that white grubs and several other soil insects are common and effective agents in the opening of infection courts for crown gall, although they are probably of little significance in actual dissemination of the pathogen.

Invasion.—Insects may perform the functions of dissemination, inoculation, and ingression in various combinations. They also may aid the plant pathogen in the invasion of the plant tissues. Leach, Orr, and Christensen (1937) have reported a case in which the insect aids the pathogen in the process of invasion of the tissues but has no part in the processes of dissemination, inoculation, or ingression. Two wood-boring beetles [*Monochamus scutellatus* (Say) and *M. notatus* (Drury)] commonly infest fallen logs of coniferous trees. By boring in a radial and tangential direction into the heartwood of pine logs, they greatly hasten the rate of invasion and decay of the heartwood by the wood-rotting fungus, *Peniophora gigantea* (see Figs. 131 and 132). The fungus spreads rapidly in the sapwood but can invade the heartwood in a radial and tangential direction only very slowly. With the aid of the insects the fungus accomplishes radial and tangential invasion, after which longitudinal invasion progresses rapidly. The fungus is disseminated and gains entrance inde-

pendently of the wood-boring insects but invades and decays the heartwood very slowly in the absence of the insects.

Preservation of the Pathogen.—In nearly all parts of the world, with the exception of some of the more humid tropical regions, there is some period of the year when it is difficult for plant pathogens to survive. The difficulty of survival is usually caused indirectly by an absence of the necessary susceptible plants on which they can persist. In the more northern latitudes, the critical period is in the winter when the susceptible plants are killed or made dormant by low temperatures. In the warmer and more arid regions, the heat and drought may be the limiting factors. Successful pathogens are those which, by means of some special adaptations, are able to survive these unfavorable periods and cause infection when conditions are again favorable.

Mere survival of the pathogen, however, is not sufficient. The pathogen must survive under conditions that provide a suitable means of reinfection. Some pathogens, capable of saprophytic growth, remain dormant in the soil or plant refuse through the unfavorable period and, when plant growth is renewed, produce inoculum that is disseminated by wind or other agencies. Some pathogens are adapted for survival in the roots or stems of perennial plants. Others survive in seed or other plant parts used for propagation. When a pathogen is largely or entirely dependent upon insect transmission, it is often adapted to survival within or on the body of the hibernating insect. As examples of some of the insects that are known to aid in the preservation of plant pathogens may be mentioned the corn flea beetles that harbor the bacteria causing bacterial wilt of corn, the pine bark beetles harboring the blue-stain fungi, the seed-corn maggot in the puparia of which the soft rot bacteria may survive, and the cucumber beetles which preserve *Erwinia tracheiphila* over winter.

THE POSSIBLE ROLE OF INSECTS IN THE ORIGIN OF NEW DISEASES AND THE EXTENSION OF OLD ONES

One other aspect of the association of insects and plant diseases generally has been overlooked. This is the possible and probable role of insects in the sudden appearance of new and destructive plant diseases. We are accustomed to thinking of plant diseases largely in terms of the pathogen, an organism of ancient lineage

that has had in common with other plants a long and slow process of evolution during which its efficiency as a pathogen also has evolved. But a disease is not an organism. It is a biological process and is the result of the interaction of two or more organisms, influenced by a large number of environmental factors. The organisms involved in the production of the disease may have evolved slowly, but the disease itself may have come into existence in a different manner. For the purpose of illustration, let us consider the Dutch elm disease, first recognized in south Holland in 1919 (Spierenburg 1922). There seems to be considerable evidence that it has spread from this center to most of the countries of Southern and Western Europe and also into the United States (May 1931, 1934). Its origin is obscure, but the general assumption seems to be that it may have been introduced into Western Europe from some other part of the world. The author has no original evidence with which to challenge this assumption but wishes to suggest a means by which the disease, in the sense of a biological process, may have originated in Western Europe within relatively recent years. The bark beetles, which constitute the principal agent of dissemination of *Ceratostomella ulmi*, were known in Europe and America for many years prior to the appearance of the Dutch elm disease (Collins 1935). At the present time, there are large areas in which the beetles occur but where the disease is absent (Felt 1934), but these areas appear to be rapidly diminishing as the result of the spread of the disease by contaminated beetles. This fact, although by no means constituting conclusive proof, would seem to indicate that the association is probably not one of long standing. *C. ulmi* is a virulent pathogen on elms; but it grows well as a saprophyte on dead wood or bark, and variant strains are known to occur (Walter 1937). The fungus can infect only through wounds, and its spores are not well adapted to wind dissemination. In the absence of a suitable insect vector, it would probably exist chiefly as a saprophyte on dead wood and perhaps be disseminated from one dead tree to another by insects inhabiting dead wood. As such it would attract no attention, and the injury caused would scarcely justify consideration as a disease. But when a virulent pathogenic strain of the fungus becomes associated with the Scolytid bark beetles, which, because of their habit of feeding on twigs of vigorous healthy trees, prove to be ideal

vectors, a very destructive disease is the result. Furthermore, the elm bark beetles, which by their nature can breed only in weakened or dying elm trees, find the association to their advantage. By transporting the spores of *C. ulmi* and introducing them into feeding wounds, they cause infection of more trees, provide themselves with an abundance of weakened or dying elms, and thereby rapidly increase in number. More vectors, more disease, more dead or weakened trees, and more vectors! A vicious circle resulting in an epiphytotic disease that threatens to destroy one of our most valuable trees! This order of events in the origin of the Dutch elm disease is purely theoretical, but it is well within the realm of possibility and deserves consideration. It is suggested here primarily to illustrate one of the many significant aspects of the association of insects with plant diseases.

There is evidence that fungi of some sort are constantly associated with most, if not all, Scolytid bark beetles. It would be interesting to know what fungi, if any, are regularly associated with the elm bark beetles in the regions where the Dutch elm disease does not occur. The author has seen no record of this having been determined.

Many insects as well as plant pathogens are, or until recently have been, relatively local in their distribution. With increased commerce and travel throughout the world, they are becoming more widespread in spite of all efforts to control them. With promiscuous intermingling of insects and pathogens, it would be surprising if new combinations and associations were not formed in which an insect and a pathogen (neither of which alone may be conspicuous or of economic importance) working together may give rise to a destructive disease. This idea, first presented by Leach (1935), has been expressed independently by Carter (1936).

The invasion of new areas by insects also may influence profoundly the distribution of certain well-known destructive diseases. Curly top of sugar beet, for example, occurs in North America only in the arid regions of the western United States. According to Carter (1930), it is limited to these regions because the insect vector (*Eutettix tenellus*) cannot thrive in the more humid regions. It has been shown by Fawcett (1927) that a disease he identified as curly top in Argentina is spread by another

leaf hopper (*Agallia stictocollis*). This insect is said to thrive in regions of relatively high humidity. If it should be introduced into the United States and prove to be a vector of curly top, there is a strong probability that the range of the disease would be greatly extended.

THE CLASSIFICATION OF THE PHENOMENA OF INSECT TRANSMISSION OF DISEASES

At various times, several authors have drawn up systems of classification of the different kinds of insect transmission of diseases. Some of these have been more complete than others; yet none has been entirely satisfactory. Obviously, all have been based on incomplete knowledge of the various phenomena, and none has claimed any degree of finality. The chief aims of such classifications are to clarify one's thoughts and to provide a better perspective of the problem. With these purposes in mind and as a matter of historical interest, some of the more important classifications are given below.

Rand and Pierce (1920) were the first to present a scheme of classification applicable to plant diseases. Their classification is as follows:

1. External transmission of parasitic microorganisms.
 - a. External dissemination and direct inoculation by the insect vectors.
 - b. External dissemination and accidental infection without direct inoculation.
2. Infection through insect wounds without dissemination by the wounding agent.
3. Internal transmission of parasitic microorganism.
 - a. Mechanical internal transmission.
 - b. Biological internal transmission.

Karl Böning (1929) criticized the classification of Rand and Pierce on the basis that the primary criterion of internal and external transmission was an artificial one and involved too much overlapping. He proposed the following simplified scheme of classification:

1. Dissemination without wounding.
2. Dissemination with wounding.
 - a. Insect obligatory.
 - b. Insect not obligatory.

A classification of the roles played by insects in the spread and development of plant diseases was devised by the author and has been used for several years in teaching a class in "Insects in relation to plant diseases." This classification is as follows:

- I. Wounding of the plant by an insect without dissemination of a pathogen.
 - A. Insect toxicogenic (psyllid yellows, mealy-bug wilt of pineapple, etc.).
 - B. Insect not toxicogenic (white grubs and crown gall).
- II. Dissemination of a pathogen by an insect without wounding the plant.
 - A. Mechanical dissemination only (sphinx moths and anther smut, flies and ergot).
 - B. Biological dissemination (rare).
 1. Insect not obligatory (flies and fire blight).
 2. Insect obligatory (theoretically possible but no examples known).
- III. Dissemination of a pathogen by an insect with wounding of the plant.
 - A. Mechanical dissemination only (bark beetles and blue stain, elm bark beetles and Dutch elm disease).
 - B. Biological dissemination.
 1. Insect not obligatory (dipterous insects and bacterial soft rot, *Dacus oleae* and olive knot).
 2. Insect obligatory (cucumber beetles and bacterial wilt of cucurbits, apple maggot and bacterial rot of apples).

Similar classifications have been made by several workers interested primarily in animal diseases. The phenomena found in insect transmission of animal diseases offer different criteria, and as a result the systems of classification are somewhat different. A few of these are given below for the purpose of comparison.

Riley and Johannsen (1932) group the ways in which arthropods may affect the health of man and other animals in the following manner:

1. Directly poisonous.
2. Parasitic, living more or less permanently on the body of their host and deriving nourishment from it.
3. Transmitters and disseminators of disease, functioning as
 - a. Simple carriers.
 - b. Direct inoculators.
 - c. Essential hosts of pathogenic organisms.

Folsom and Wardle (1934) recognize the following types of association between insects and diseases of man and animal:

1. Toxic inoculation.
 - a. Venomous biters.
 - b. Venomous stingers.
 - c. Irritant caterpillars.
2. Entomiasis (disease caused by actual presence of an insect in the host body).
3. Mechanical transmission.
4. Cyclical transmission (insect an essential alternate host for a part of the life cycle of the microorganisms).

Huff in 1931 published "A proposed classification of disease transmission by arthropods." The essence of this classification is given below.

1. Biological.
 - a. Cyclopropagative (organisms undergo cyclical change and multiply within the vector).
 - b. Cyclodevelopmental (organisms undergo cyclical change but do not multiply within the vector).
 - c. Propagative (the organisms undergo no cyclical change but multiply within the vector).
2. Mechanical (organism undergoes neither cyclical change nor multiplication within the vector).

Certain factors must be kept in mind in comparing these different schemes of classification. The term "biological" has been used with many different connotations. Strictly speaking, any kind of insect transmission is biological, for living agents are involved. The term is generally used in a restricted sense in contrast with the term "mechanical," but here too its use has not been consistent. Biological transmission, as used in plant pathology, generally implies that the pathogen lives and multiplies in the body of the vector. When used in reference to virus diseases, it has a slightly different connotation. In plant pathology, biological transmission is not synonymous with cyclical transmission. Cyclical transmission occurs only when the vector serves as an essential alternate host for the sexual stage of the life cycle of the pathogen. No such relationship is known in plant pathology, with the possible exception of *Phytomonas davidi* which parasitizes species of *Euphorbia* and is transmitted by *Stenocephalus agilis* Scop. According to Franca (1920), this protozoan undergoes a simple form of cyclic change in the body of its insect vector. Large forms are found commonly in the intestine; but smaller individuals appear, presumably after

a sexual process, and these invade the salivary glands and are introduced into the plant in the feeding process.

THE BIOLOGIC AND EVOLUTIONARY SIGNIFICANCE OF THE ASSOCIATION OF INSECTS AND PLANT PATHOGENS

Research workers in phytopathology have, as a rule, considered insects merely as one of several agents that disseminate inoculum, to be disposed of in the ordinary routine study of a disease. It is the opinion of the author that the relation of insects to plant diseases is too important and too complex to be handled in such a summary manner. The association of insects with plant pathogens is, for the most part, not a mere matter of chance but rather a highly organized relationship which, in many cases, may have arisen out of a close association of insects and microorganisms over a period of 50 million years or more. It is a definite biologic phenomenon of evolutionary significance and has broad biologic and economic implications. As such it deserves more than routine attention. It merits the same type of sustained and coordinated thought and effort that has been given to other phytopathological problems of fundamental nature.

As a biologic phenomenon the association of insects and plant pathogens is in many respects similar to that of entomophily, *i.e.*, insect pollination. All students of botany or entomology are familiar with the remarkable evolutionary adaptations of insects and flowers. It will not be necessary here to describe the various details of these adaptations. Along with the morphological adaptations on the part of both insects and flowers, there has been built up a constant and regular association of insects and plants that is mutually beneficial to both parties of the association. Let us consider how this association came about. It is agreed by most, if not all, students of plants that our most primitive flowering land plants were wind-pollinated. Pollination was a matter of chance, and only a very few pollen grains reached the proper stigmatic surface. This necessitated the production of enormous quantities of pollen and obviously resulted in a tremendous waste of materials. But in one fortunate period long ago, insects discovered the food value of pollen and perhaps the associated secretion of nectar and began to make regular visits to those plants producing it. In this way, the pollen was transported with very little loss to the stigmatic surface of the proper

plant. This association had a survival value for both insects and flowering plants and consequently has persisted throughout the ages, developing to a state of very high efficiency. A little reflection will show that the principle underlying this association is essentially the same as that underlying insect dissemination of plant pathogens. Some pathogens are disseminated only by wind, in which case large quantities of inoculum are produced but a relatively small part of it reaches an infection court and

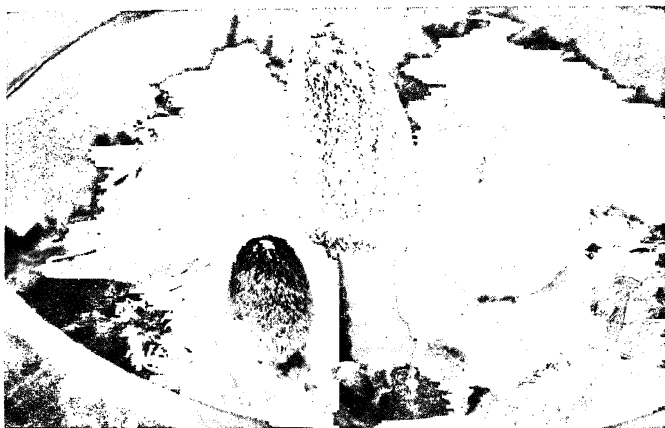


FIG. 42.—A sexual fruiting body of *Coprinus lagopus*. Basidiospores of two sexes are produced on the gills of the mushroom. The mycelium arising from a single basidiospore cannot produce another mushroom until it has been fertilized by fusion with a mycelium of the opposite sex. (Photograph by Dr. W. F. Hanna.)

survives. Other pathogens have become associated with insects which transport them or their spores to the required infection court. This is accomplished with a minimum of waste and a maximum of efficiency, especially when the insects provide also a means of ingress and of protection of the pathogen against the elements. This type of association, like insect pollination, has a survival value, and numerous cases have persisted with remarkably complex adaptations on the part of both insect and pathogen. Survival value does not predicate benefits by both insect and pathogen, but there are numerous cases of striking mutual benefit and interdependence. As examples may be mentioned the association between the blue stain of fungi and bark beetles

or that between the bacteria causing soft rot and the seed-corn maggot.

It would not be logical, however, to drop the comparison here, for there are other important factors that have influenced the complicated adaptations associated with entomophily. With the development of insect pollination, the nature of the process led to an increase in cross-pollination in comparison with self-pollination. Cross-pollination increased the frequency of hybridization, and this in turn stimulated variation, a primary factor in evolution. Thus, insect pollination, by stimulating hybridization and variation, has speeded up the development of entomophily.

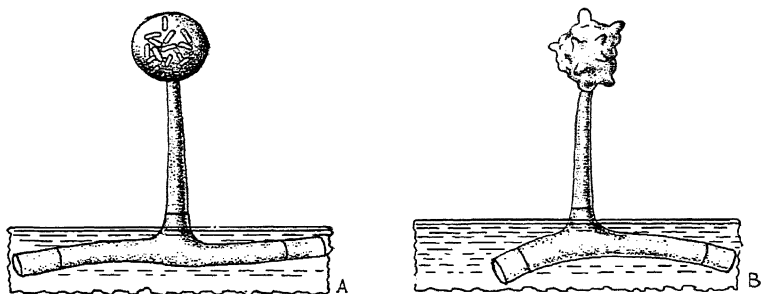


FIG. 43.—Conidiophores of *Coprinus lagopus*. Note that the spores are borne in drops of a sticky solution. A, sporophore in a moist atmosphere; B, the same after exposure to dry air. (After Brodie.)

ily itself. This factor, so important in evolution, is apparently lacking in many of the associations of insects and plant pathogens and may account for the fact that the latter association is less obvious than entomophily.

But recent investigations have shown us that the factor of hybridization cannot be removed entirely from the picture. With the discovery of heterothallism in fungi by Blakeslee (1904) and its further elucidation by Kniep (1928), Buller (1931), Stakman *et al.* (1930), and others, the significance of hybridization in the evolution of fungi is more clearly discernible.

Brodie (1931) has shown that flies are agents of "diploidization" in the fungus *Coprinus lagopus*, a process fundamentally analogous to insect pollination of higher plants. *Coprinus lagopus*, a mushroom commonly found on horse dung (Fig. 42), is a heterothallic fungus. Each basidiospore formed on the

"inky-cap" mushroom, upon germination, forms a homosexual mycelium that is unable to reproduce the mushroom stage until it has been fertilized or diploidized by fusion with a mycelium of the opposite or complementary sex. Each of these homosexual mycelia produces conidia in drops of a sticky solution that prevents them from being blown around by air currents but that is attractive to flies (Fig. 43). Flies, by feeding on these droplets, transport the conidia from one mycelium to another (Fig. 44). The conidia promptly germinate and fuse with mycelia of opposite sex; diploidization results, followed by the formation of the mushroom stage. The spores may pass uninjured through the



FIG. 44.—A diagram illustrating role of flies in the diploidization of the mycelium of *Coprinus lagopus*. Portions of two colonies of the fungus growing on agar are shown. Each colony originated from a single basidiospore, one of one sex (+) and one of the opposite sex (-). Both mycelia are producing conidia in the usual drops of liquid. The fly in feeding has moved from one colony to the other taking with it spores that will germinate and fuse with the mycelium of the opposite sex, resulting in diploidization (fertilization). (After Brodie.)

digestive system of the flies, and drops of excrement usually contain spores of both sexes. Dowding (1931) has pointed out a comparable association between *Ascobolus stercorarius* (Bull.) Schröt. and mites and flies.

A similar condition in the rust fungi has been revealed by the work of Craigie (1931) on *Puccinia helianthi* Schw. and *P. graminis* Pers. The rusts are heterothallic fungi. Reduction division occurs in the germinating teliospore (Fig. 45), and on germination and infection the sporidia give rise to haploid mycelia. Pycnia (Figs. 46 and 47) are produced on the haploid mycelium, but aeciospores, being dicaryotic, are formed only after diploidization. The process of diploidization has been described by Craigie (1931), Buller (1938), and others. It involves the transfer of

pycniospores from one pycnium to another of the opposite sex. Here the pycniospores germinate and fuse with a receptive mycelium through the "flexuous hyphae" first described by

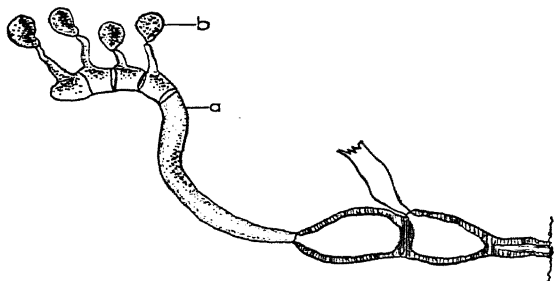


FIG. 45.—A germinating teliospore of *Puccinia graminus*. The complete promycelium (a) of only one cell is shown. Reduction division takes place in the promycelium, and each sporidium (b) is in the haploid condition.

Craigie (1933) (Figs. 48 and 49). The transfer of the pycniospores is often accomplished through the agency of insects. The pycniospores are exuded from the pycnium in drops of a fragrant, sugary solution that makes them poorly adapted for dissemina-

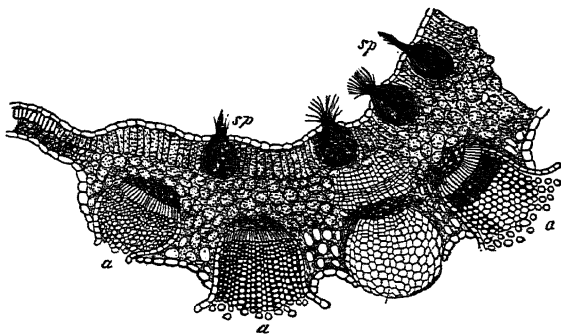


FIG. 46.—A section through a barberry leaf infected with *Puccinia graminus* showing the pycnia (sp) and aecia (a). The aecia do not develop until the mycelium has been diploidized by the fusion of a pycniospore with a flexuous hypha as shown in Fig. 48. (Modified, after Sachs.)

tion by wind but well adapted to insect transfer (Fig. 50). Many species of rusts have developed special characters that attract insects and ensure the necessary interchange of sporidia. Among

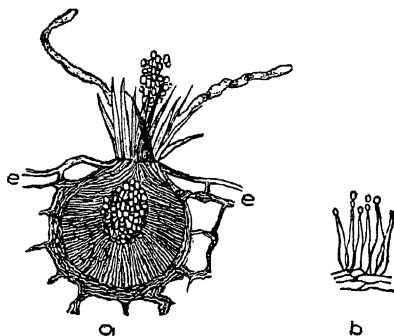


FIG. 47.—A diagrammatic sketch of a section through a pycnium of *Puccinia graminus*. *a*, pycniospores oozing from the mouth of the pycnium surrounded by the spinelike setae and two of the flexuous hyphae; *b*, an enlarged view of a section of the pycnium wall showing how the pycniospores are borne; *c*, epidermis of the barberry leaf. The pycniospores are functional gametes, and diploidization is effected when a pycniospore is transported to a pycnium of the opposite sex where it germinates and fuses with a flexuous hypha as shown in Fig. 48. The sugary exudate is not shown in this sketch. (Modified after De Bary.)



FIG. 48.—The fusion of a germinated pycniospore with a flexuous hypha of a pycnium of *Puccinia helianthi*. The nucleus of the pycniospore has passed into the flexuous hypha. By repeated division and subsequent migration of the daughter nucleus into the adjacent cell, the mycelium of the pustule is rapidly diploidized. Aecium formation soon follows. Approx. 1,500 \times . (After Craigie.)

these special adaptations may be mentioned the sugary solution in which the pycniospores and flexuous hyphae are produced, the bright orange color of the pycnia, and the distinct fragrance of the sugary exudate.

Flies, attracted by the drops of fragrant, sugary solution formed on the bright orange-colored pycnia, effectively transport the pycniospores from one pycnium to the other. Thus, insect transportation of pycniospores results in diploidization and

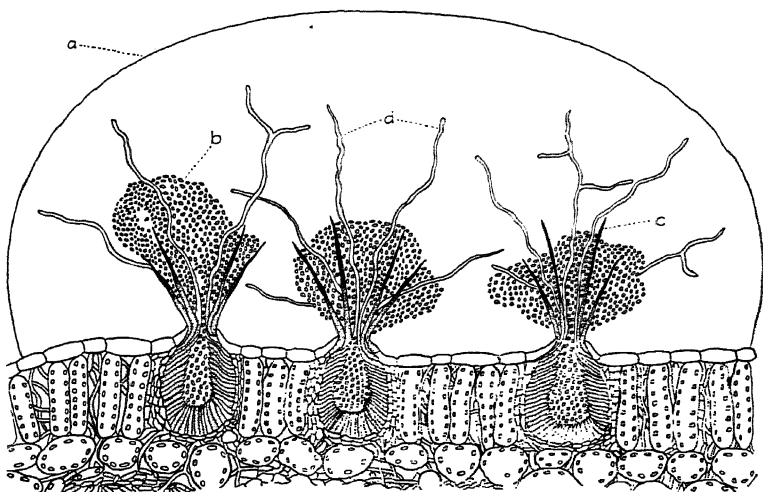


FIG. 49.—A diagrammatic representation of pycnia of *Puccinia graminus* showing the flexuous hyphae with which pycniospores of the opposite sex fuse. The curved line (a) represents the surface of the drop of sugary exudate; b, pycniospores; c, setae; d, flexuous hyphae. (After Buller.)

increases the chances of hybridization and variation. The relationship is quite comparable to insect pollination of flowering plants. The large number of physiologic forms that occur in this pathogen can be explained to some extent by the hybridization that occurs on the barberry (Stakman *et al.* 1930). It is probably not a mere coincidence that such a specialized adaptation for insect "pollination" of a fungus, with resulting hybridization, should be found in one of the most highly developed plant pathogens and one in which physiological specialization has reached such a high state of development.

The association of insects with the pycnia of rust fungi was observed and discussed at length by Plowright as early as 1889, over forty years before the function of the pycnia was discovered. Plowright observed 135 species of insects, including 31 Coleoptera, 32 Hymenoptera, 64 Diptera, and 8 Hemiptera feeding on the nectar. He recognized specific adaptations of the rusts for insect attraction, listing the following six: (1) the constant pro-



FIG. 50.—A leaf of the common barberry infected with *Puccinia graminis*, showing four groups of pycnia with the droplets of sugary solution in which the pycniospores are borne. In natural color, the light areas are brilliant orange. Notice the reflection of the light by each droplet. The sugary exudate has a faint but distinct fragrance. The entire fructification appears to be designed for the attraction of insects.

duction of a sugary exudate in which the pycniospores are borne, (2) a definite fragrance that is associated with the pycnia of many species of rusts, (3) the bright orange color of the pycnia, (4) the abnormally erect position of affected leaves of some plant species, (5) the lighter color of pycnia-bearing leaves as compared with normal leaves of some plants, and (6) the dark spots contrasting with a lighter background characteristic of the pycnia of some rust species. This remarkable adaptation for the attraction of

insects is strikingly similar to the adaptations of flowering plants for the same purpose. It is very probable that diploidization in the rusts, through the agency of insect "pollination," has had a directing influence in the evolution of this interesting and economically important group of fungi.

The extent to which insects are involved in the mechanics of diploidization in other fungi is a problem for future study, because it has not yet received much attention. It is of interest to note that Drayton (1934), who has described the sexual mechanism of *Sclerotinia gladioli* (Massey) Drayton, is of the opinion that insects may play a part in the sexual reproduction of that fungus. He points out that the microconidia are borne in a mucilaginous matrix, the sticky nature of which would "allow the sporodochia to adhere to the bodies of soil-inhabiting animals or insects, and so be carried through the soil, possibly to receptive bodies of a compatible thallus."

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CHAPTER V

PLANT DISEASES CAUSED BY TOXICOGENIC INSECTS

It has become well recognized in recent years that a number of very destructive diseases of plants are caused by toxic substances introduced into the tissues of the plants by sucking insects. These diseases often bring about greater destruction than do the more direct injuries caused by chewing insects. Because of the indirect nature of the injury, the causal relationship of the insect frequently has been overlooked, and the true nature of the disease has escaped discovery. Relatively little work has been done on diseases of this type, and the nature of the toxic substances and the methods of their action are poorly understood. It is sometimes difficult to distinguish between pathological conditions resulting from purely mechanical injuries that may disturb the physiology of the plant and those resulting from definite toxic substances. For example, the well-known hopperburn of potato and other plants is thought by some to be caused by a diffusible toxic substance introduced by the hopper, while others maintain that the mechanical injury caused by feeding is sufficient to account for the ill effects. These differences of opinion will continue until we know more about the physiology of the diseased plants and of the insects involved in producing the diseases.

Carter (1936a) has introduced two new terms to describe insects that may cause diseases by the injection of toxic substances. He observes that, although the diseases caused by the insects may differ widely, they all have certain characteristics in common. All are characterized by specific toxic effects, and the capacity to produce these is inherent in the insects concerned. Species of insects producing the toxic substances were termed "toxicogenic." Since, as was shown by Carter (1936b), certain individuals of a species may, under some conditions, lose the ability to secrete the toxic substance, it was desirable to have a term to express the active toxic stage of a toxicogenic insect. For this he proposed the term "toxiferous."

A species capable of secreting a phytotoxic substance is called a "toxicogenic" species. A toxicogenic insect is "toxiniferous" only in those stages of development when it can secrete the toxic materials. In a later paper, the same author (1939) presented a general review of plant injuries caused by insect toxins in which he refers to this group of diseases as "toxemias," "phytotoxemias," or "toxicoses." Whatever term may be used to designate these plant injuries, they constitute a group of economically important diseases that are very imperfectly known.

Diseases caused by toxicogenic insects may resemble virus diseases and are sometimes confused with them. The systemic phytotoxic effects found in such diseases as psyllid yellows of potatoes and mealy-bug wilt of pineapples resemble virus diseases in many respects. It is difficult without extensive experiments to determine to which category a disease belongs, and as a result the diseases are often confused in the mind of the student. Carter (1936c) recognized this situation and has pointed out some of the more significant differences between the two classes of disease. The essential differences are based largely on the inability of the toxic secretion of the toxicogenic insect to reproduce itself in the plant tissues. The following table of comparison is based on that constructed by Carter but has been slightly modified and is given in a more generalized form.

Diseases Caused by Toxicogenic Insects	Virus Diseases
1. Caused by a phytotoxic secretion of an insect	1. Caused by a virus
2. Toxic secretion is nonreproductive in plant	2. Virus is reproductive in plant
3. Recovery common	3. Recovery rare
4. Degree of injury related to the length of time, and number of insects feeding	4. Injury not directly related to length of time and number of insects feeding
5. Capacity to produce inherent in insect	5. Capacity of insect to produce acquired only by feeding
6. Disease not perpetuated by vegetative propagation or transmitted by grafting	6. Disease can be perpetuated by vegetative propagation and transmitted by grafting
7. Control of disease in proportion to insect control	7. Control of disease usually not in proportion to insect control

If the viruses prove to be of the nature of autocatalytic proteins, as indicated by the work of Stanley (1935) and others on tobacco mosaic, the similarities between these two groups of diseases will be greater still. The demonstration by Fukushi (1933, 1935) that the virus of rice dwarf is congenitally transmitted through the eggs of the vector has nullified to some extent item 5. The matter of reproduction on the part of the disease-inciting substance appears to be the most significant of the recognized differences. Even this distinction may have its exceptions. Severin and Haasis (1934) have demonstrated the transmission of the California strain of aster yellows to potato by the leaf hopper [*Macrostelus divisus* (Uhl.)], but they were unable to recover the virus from infected potato plants. Leaf hoppers did not become viruliferous after prolonged feeding upon infected potato plants or after feeding upon the cut surface of tubers from plants that had shown symptoms of yellows. If these negative results are confirmed, showing that a virus will cause distinct symptoms on a plant but that the virus cannot be recovered from the infected plant or its progeny, it will be still more difficult to place borderline cases in one category or the other. Furthermore, it will be necessary to reinvestigate some of the diseases that resemble virus diseases but that are now interpreted as toxemias. For the best understanding of the nature of the disease caused by toxicogenic insects, it will be advisable to consider in some detail a few of the better known examples.

Stigmonose.—A. F. Woods (1900) was one of the first to recognize a definite plant disease as being caused by the indirect effects of insect feeding. Woods investigated a leaf-spotting disease of carnations that previously had been diagnosed as a bacterial disease (Arthur and Bolley 1896) and demonstrated that aphids, and not bacteria, were the actual cause of the trouble. Woods concluded that "the insect injects some irritating substance of an acid or enzymic nature into the wound, that this substance causes the increase of oxidizing enzymes in the cells which it reaches, and that these enzymes interfere with the nutrition of the cell by destroying the chlorophyll and setting up other changes which finally result in death." Woods applied the term "stigmonose" or puncture disease to this abnormality. The term has since been used to designate diseases on many plants that are charac-

terized by local lesions caused by insect punctures as, for example, those often found on apples resulting from the feeding activities of the rosy apple aphid.

Since most phytophagous insects puncture plants in the process of feeding, the number of species that cause local lesions is extremely large. The type of injury varies from scarcely observable flecks to prominent and very characteristic necrotic lesions. It would seem unnecessary to discuss many of them here. A more inclusive account of this group of insect injuries has been presented by Carter (1939).

Hopperburn of Potato and Other Plants.—Hopperburn is of particular interest because its history illustrates the extent to which the real cause of a disease of this kind may be overlooked and misinterpreted. It illustrates also the difficulty in distinguishing between the effects of simple mechanical injury and those of a toxic substance. The belated discovery of the true cause of the disease served in no small measure to call to the attention of both pathologists and entomologists the possible significance of toxicogenic insects in the production of plant diseases.

In 1895, L. R. Jones, working in Vermont, described a disease of potato characterized by necrosis of the tips and margins of the leaves and called it "tipburn." He distinguished it from the fungus diseases, early blight and late blight, and concluded that it was due to excessive transpiration under the influence of high temperature, low humidity, and bright sunlight. In the following year, Osborn (1896), working in Iowa, called attention to the leaf hopper (*Empoasca fabae* Harris) as a potato pest and briefly described the injury caused by it. The extent of this injury was not fully recognized by entomologists, and plant pathologists usually confused the injury with the physiological tipburn described by Jones. E. D. Ball (1918), working in Wisconsin, was the first to distinguish clearly between the two types of injury. He demonstrated the nature and extent of the leaf-hopper injury and applied the term "hopperburn" to it. Ball (1919a, 1919b) clearly differentiated the symptoms of hopperburn from those of tipburn, pointing out that in the latter the mesophyll tissues between the veins were the first to die as a result of excessive transpiration, while in hopperburn the veins were affected first, and the injury later spread to the mesophyll that

was served by the veins (Figs. 51 and 52). Ball also expressed the opinion that the death of the tissues in hopperburn was due

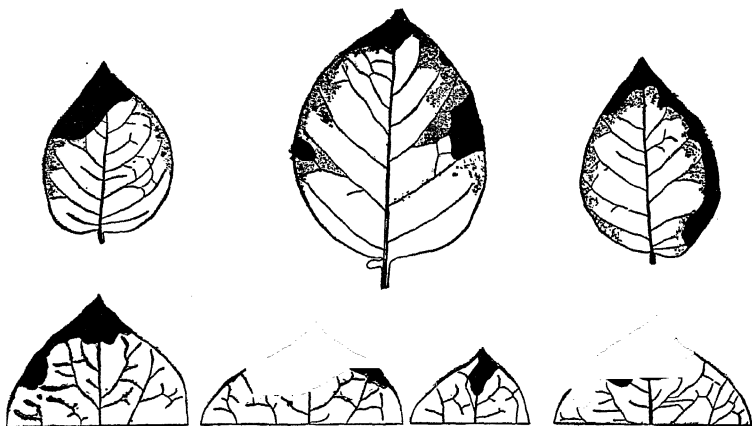


FIG. 51.—Potato leaves affected with physiological tipburn. Note that the necrotic or burned areas are not greatly influenced by the location of the veins. (After Luitman.)

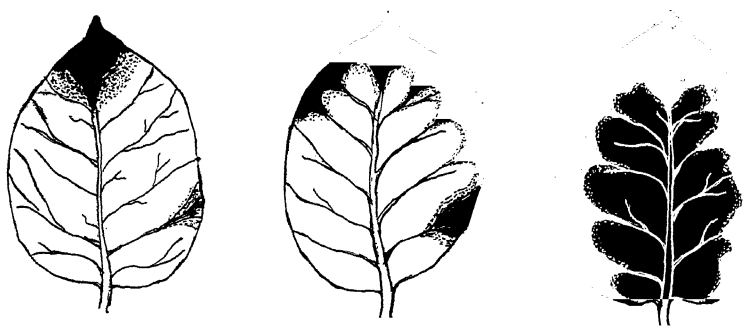


FIG. 52.—Potato leaves affected with hopperburn. Diagrammatic sketch to illustrate how the burning has its origin at a point on the vein where the hoppers have fed. The necrotic areas tend to form a triangle with its apex at the feeding point on the vein and its base at the leaf margin.

to some specific toxic substance transmitted by the insect. Later, Ball and Fenton (1920) suggested the substitution of “hopperburn” for “potato tipburn” because the name “covers practically all that has formerly been designated as tipburn on

this plant." Lutman (1919, 1922), however, has shown clearly that tipburn is a distinct disease and may be very destructive in years when leaf hoppers are absent or in regions where they do not occur. Furthermore, as pointed out in 1922 by Leach, the necrosis of leaf tissue is only one stage of the injury caused by the hoppers. When they feed on the young leaves, there is a pronounced retardation of development characterized by shorten-



FIG. 53.—The leaf-curling effect of leaf-hopper feeding on potatoes. Leaves B and D from plants infested with leaf hoppers in a cage. Note the curled leaves, shortened internodes, and smaller size when compared with leaves from leaf-hopper-free plants (A and C). The leaves are of comparable age and from comparable locations on the plants. No necrosis or burning is present on either set of leaves.

ing of the petioles and crowding of the leaflets. There is also a downward curling of the mid-veins accompanied by an upward folding of the leaf along the mid-vein (Fig. 53). The tissues thus affected are noticeably more brittle than normal ones. These symptoms never result from excessive transpiration. It is evident that there are two distinctly different diseases with different causes, and they should not be confused.

The potato leaf hopper (*E. fabae* Harris) (Fig. 54) is generally distributed throughout North America and feeds upon a wide

variety of plants. It overwinters in the adult stage under leaves, weeds, and other debris. The females become active in early spring and feed on apple trees, beans, alfalfa, and a number of weeds. The males do not come out of hibernation until later. During June, the females migrate to potatoes, beans, alfalfa, and other host plants and are joined by overwintered males. Egg

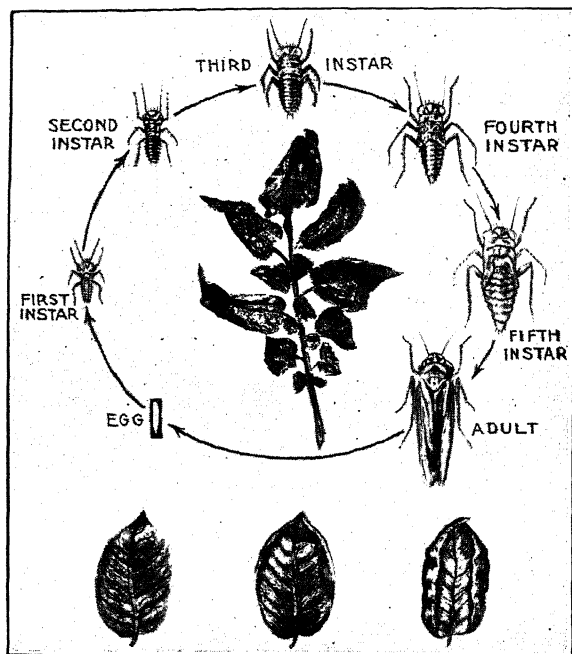


FIG. 54.—The potato leaf hopper (*Empoasca fabae*) and potato leaves affected with hopperburn. (After Metcalf and Flint.)

laying begins soon, the eggs being inserted into the mid-rib and leaf veins, chiefly on the undersurface. The eggs are only $\frac{1}{24}$ inch in length and are difficult to detect in the tissues. They hatch after about 1 week, and the nymphs complete their development, comprising five instars, in 2 to 4 weeks, depending upon the temperature. The nymphs begin feeding immediately, and the characteristic hopperburn soon appears. The nymphs are wingless and unless disturbed complete their development on the

plant on which they are hatched. The winged adults are more active and migrate to other plants when those on which they are feeding mature. The various broods overlap extensively, and there are two to four broods each year.

Following Ball's report on hopperburn of potato, similar injuries caused by the same insect on alfalfa, clover, apple, and other host plants were described by other workers who investigated more thoroughly the nature of the injury. The work of Eyer (1922), Fenton and Hartzell (1923), Jones and Granovsky (1927), Granovsky (1926, 1928, 1930), Monteith and Hollowell (1929), and Poos (1929) led to the conclusion that the direct cause of the injury was a diffusible toxic substance or enzyme injected into the plant by the leaf hopper. In 1930, Granovsky reported that microchemical tests revealed a greater accumulation of starch grains and sugars in affected tissues. There was also a clogging of vascular bundles and complete disorganization of the phloem region of severely injured tissue, phenomena held to be due to enzymic secretions by leaf hoppers in the course of feeding. Later investigations by Smith and Poos (1931), Smith (1933), and Johnson (1934) have verified the histological changes reported by Granovsky, but these authors consider it improbable that the leaf hopper injects a toxic substance. They suggest that the overaccumulation of carbohydrate products of photosynthesis above the point of feeding, as a result of the mechanical injury of the feeding punctures, is sufficient to cause the symptoms usually attributed to a diffusible toxic substance.

Empoasca fabae, unlike a number of closely related, but less injurious species, feeds almost exclusively on the vascular tissues, the mouth parts usually penetrating the phloem elements. The line of puncture becomes surrounded by a sheath of proteinlike substance shown by Smith and Poos (1931) to be largely of insect origin. The disorganization of cells some distance removed from the line of penetration would indicate that the secretions of the insect were to some extent toxic, at least locally. Johnson (1934) was unable to duplicate the disruption and plugging of the phloem tissues by repeated puncturing of a restricted region of leaf petioles with a fine glass needle. From this fact, it would seem highly improbable that the mechanical injury alone would so completely disorganize the phloem tissue. It would seem that a certain amount of a toxic substance must be injected by the

insect although the major symptoms and the ultimate death of the tissues beyond the point of puncture may be the direct result of the accumulation of photosynthetic materials brought about by the clogging of the phloem. Toxicity is, at best, a poorly defined concept, because the toxic substance may exert its effect entirely through abnormal physiological reactions.

There are other species of leaf hopper that feed upon the vascular bundles but that do not cause injury as pronounced as that caused by *E. fabae*. The difference is probably due to an unequal degree of toxicity of the saliva of the various species. It is evident that neither a virus nor a widely diffusible toxic substance is involved in the production of hopperburn; for the disease is not systemic, and the amount of injury is always in direct proportion to the number of leaf hoppers present.

The recognition of this disease and its relation to leaf-hopper infestation has led to important discoveries in the principles of insect and plant-disease control. While hopperburn was still confused with tipburn caused by excessive transpiration, experiments had shown that fairly effective control was obtained by spraying the plants with Bordeaux mixture, although it was known that Bordeaux mixture usually stimulated transpiration. This apparent paradox was explained by further experiments, made after the role of the insects had been discovered, which showed that the leaf hoppers were fairly well controlled by the Bordeaux mixture. As the leaf hoppers are sucking insects and Bordeaux mixture is not a strong contact insecticide, it was difficult to account for the control. It was concluded by Fluke (1919), Kotila (1922), and others that the Bordeaux mixture acted on the leaf hopper mainly as a repellent, discouraging oviposition. De Long, Reid, and Darley (1930a, 1930b), however, have shown that the leaves absorb enough copper from the spray to be toxic to the leaf hoppers, especially the nymphs, as they feed upon the cell contents. Although it has been known for some time that copper from sprays was absorbed in small quantities without injury to the cells, the effects of the absorbed copper on sucking insects had never been extensively investigated. This is essentially a new principle of insect control, and its possibilities in the control of sucking insects have not been fully exploited.

The influence of the degree of pubescence of certain varieties and strains of clover and soy beans on hopperburn as demon-

strated by Hollowell, Monteith, and Flint (1927), Granovsky (1928), Pieters (1929), Jewett (1929), Poos and Smith (1931), and Johnson and Hollowell (1935) is of much interest. These workers show that the pubescent varieties are more resistant to leaf-hopper injury than the glabrous ones (Figs. 55 and 56). Pieters



FIG. 55.—Stems of two varieties of red clover and a mature leaf hopper showing the differences in pubescence and the relative size of the trichomes and the insect. Left, a stem from a very susceptible Italian red clover. Right, a stem of the relatively resistant Tennessee red clover. The resistance is attributed to dislike of the dense pubescence by the leaf hoppers. Insert, a mature leaf hopper photographed on the same scale to show relative size of the insect and the trichomes. Approx. 4×. (After Monteith and Hollowell.)

has suggested that the predominance of the pubescent varieties of red clover in America can be explained by the unconscious selection of these types because of their resistance to the leaf hopper. The preference of the female hoppers for the glabrous tissue for oviposition is given as the factor responsible for the resistant, or escaping, character, although some workers believe other factors are involved.

The resistance of certain varieties of apple trees to injury by the leaf hopper also has been explained on this basis by Schoene and Underhill (1937) who showed that the susceptibility to hopperburn varied inversely with the amount of pubescence of the leaves of the different apple varieties. Varieties having foliage with scant pubescence (Albermarle Pippin, Bonum, and Early Harvest) were subject to serious injury while those varieties with densely pubescent foliage (Stayman Winesap, Winesap, and Delicious) were injured only slightly.

Hopperburn, or yellow top, of alfalfa was long confused with the effects of fungus leaf spots, and little or no effort was made to



FIG. 56.—The resistance of pubescent types of soy beans to hopperburn. The two large plants are highly pubescent and because of this fact have escaped the injury suffered by the remaining glabrous plants. (After Johnson and Hollowell.)

control the leaf hoppers; but following the recognition of the part played by leaf hoppers (Jones and Granovsky 1927), the injury has been reduced considerably by delaying the early cuttings of alfalfa until just after the height of the egg-laying period. By delaying the cutting until most of the eggs are deposited, a high percentage of the eggs will be destroyed, and the later cuttings will be relatively free from hoppers (Graber and Sprague 1933, 1935; Searles 1934; and Jewett 1934).

Toxicogenic Capsid Bugs and Effects on Plant Tissues.—Kenneth M. Smith (1920) has studied the nature of the injury to apples caused by a toxicogenic "capsid" bug. Of five species of capsids that normally feed on the fruit and leaves of apple trees in England, only one species (*Pleisicoris rugicollis*) is responsible

for appreciable damage. This species causes the death of the tissues surrounding each feeding puncture; and when the insects feed on young apple fruits, the mature fruits become much distorted and severely russeted. Smith showed definitely that the injury was caused by the secretion of a toxic substance in the saliva of the insect. When the salivary glands of the different species were dissected out and placed on the cut surface of apple tissue, much of the tissue was killed around the glands of *P. rugicollis*, while that adjacent to the glands taken from the remaining species was not injured. Smith was unable to reproduce the injury by mechanical means. Moreover, the fact that other species of capsid bugs, feeding in the same way on the same kinds of tissue, produced no such injury added weight to the evidence of the toxicogenic nature of *P. rugicollis*.

More recently, Leach and Smee (1933) and Leach (1935) have shown that a stem canker of tea and mangoes, an angular leaf spot, a fruit scab, and a fruit rot of mangoes are caused by the bug *Helopeltis bergrothi* Reut. The various lesions produced by the feeding punctures of the insect simulate to a marked degree those caused by many pathogenic fungi and bacteria, and, according to Leach, the injuries have frequently been diagnosed as fungus diseases. These authors were unable to find any microorganisms associated with the lesions and demonstrated the noninfectious nature of the trouble. They conclude that the necrosis of the tissues is due to the injection by the insect of some irritant that diffuses through the cells from which the sap is sucked.

Froghopper Blight of Sugar Cane.—The Cercopid *Thomaspis saccharina* Dist. causes a destructive disease of sugar cane known as "froghopper blight." Primary symptoms consist of pale chlorotic lesions at the point of puncture on a leaf. The lesion gradually enlarges, assumes a pink color, and later becomes brown and necrotic. There is a shortening of internodes resulting in a stunted plant. Adventitious buds and roots are formed in excessive amounts near the base of the plants. An increase in acidity of the cell sap is usually a characteristic of the affected plants. Recovery may take place if growing conditions are favorable after the insects leave the plant.

The disease has been studied extensively by Williams (1921) and Withycombe (1926). These workers conclude that the

disease is caused entirely by the feeding of the froghopper and not by fungi, bacteria, or viruses transmitted by the insect. Injury results partly from the withdrawal of large quantities of moisture from the tissues and partly from the injurious effects of the salivary secretions of the insect. The latter is accomplished in part by diastatic and oxidizing enzymes. The rate of respiration is increased locally, and translocation is inhibited, resulting in a general upset in the metabolic equilibrium of the plant.

The severity of the injury varies greatly with those factors of the environment which influence the general physiology of the plant. Since the froghoppers oviposit in the soil, the soil reaction and soil type are considered to influence the prevalence of the insects.

Mealy-bug Wilt of Pineapple.—This disease has been known in Hawaii since 1910, but its cause has been known only since 1931. It has been reported also in Jamaica and in Central America (Carter 1934). It is extremely destructive and is considered the most important cause of pineapple failure in Hawaii. Illingworth (1931) was the first to show that the disease was in some way associated with the attack of the mealy bug [*Pseudococcus brevipes* (Ckl.)]. Since 1931, it has been studied intensively by Carter (1932, 1933, 1935*a*, 1935*b*, 1936, 1937) and Carter and Schmidt (1935), who have added much to our knowledge of the nature of the disease. They have shown that the mealy bug (*P. brevipes*) is a toxicogenic insect and that the disease is caused by a toxic substance injected into the plant by the mealy bugs in the act of feeding.

The symptoms of mealy-bug wilt are variable, being influenced by the size of the initial population of mealy bugs, the age and vigor of the plant, and other variable factors. Carter (1933*a*) distinguishes two distinct types of wilt, quick wilt and slow wilt. Quick wilt occurs primarily on young plants and usually follows the attack of an initial population of 50 or more mealy bugs (Fig. 57). A short period of feeding is sufficient to produce the disease, but it requires about two months for the appearance of the typical symptoms. Complete establishment of the colony of mealy bugs is not necessary for the production of quick wilt. The leaves of affected plants become flaccid, droop, and turn pale, the color varying from very light dull green to pale yellow

or pink. On older plants, some of the leaves take on a conspicuous red color. The tips of the leaves become brown and dry. The meristematic tissues are not killed, and cuttings made from affected plants grow normally. Apparent recovery from quick wilt is common, but the fruits formed are small and of little commercial value.

Slow wilt develops on older plants when a large colony of mealy bugs is built up from a small initial population. Unlike quick wilt, slow wilt is always fatal. The leaves wilt but,

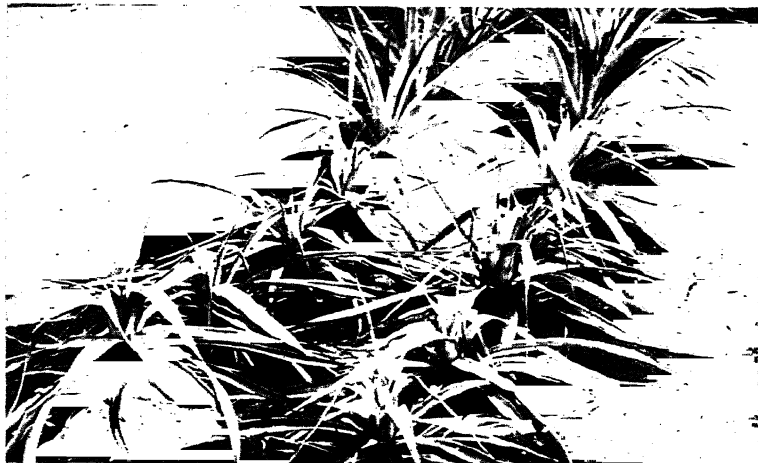


FIG. 57.—Mealy-bug wilt of pineapple. “Quick wilt.” This type most frequently occurs on young plants after the attack by a colony of 50 or more mealy bugs. Plants often recover from quick wilt. “Slow wilt” occurs on older plants after a colony of mealy bugs has been built up slowly from a small initial population. Slow wilt is always fatal. (After Carter.)

because they are older, droop less than those affected with quick wilt. The tip becomes necrotic, but the yellow and pink color characteristic of quick wilt is absent. The roots collapse and are invaded by various secondary organisms that contribute to the final destruction of the plant. Green spots associated with the feeding punctures of the mealy bugs are often found on plants affected with each type of wilt. The wilt is usually more severe when the green spots are present, but Carter considers the production of green spots a distinct disease and not a symptom of mealy-bug wilt.

The mealy bug (*P. brevipes*) (Fig. 58) is a member of the homopterous family Coccidae. In Hawaii, it is found on many kinds of plant, its principal hosts being pineapple, banana, sisal, and several species of grasses, including *Panicum barbinode* and *Tricholaena rosea* Nees. The insect is parthenogenetic and ovoviviparous, requiring about two months to complete its life cycle. The insects' bodies are covered with a characteristic



FIG. 58.—A colony of pineapple mealy bugs (*Pseudococcus brevipes*) on a pineapple plant.

white waxy secretion, and they also secrete honeydew. The mealy bugs are constantly attended by two species of ants [*Pheidole megacephala* (Fabr.) and *Solenopsis germinata* Fabr. var. *rufa* Jerdon], and colonies will not develop normally when the ants are excluded.

Based on the results of a long series of experiments, Carter's conclusions are that mealy-bug wilt is caused by a phytotoxic secretion that is diffusible but incapable of reproduction in the plant. The failure of quick-wilt symptoms to develop when colonies were built up slowly from a small initial population

was interpreted as the result of an antitoxic reaction on the part of the host. Evidence was presented to show that the toxicity of the secretions varied with the kind and condition of the host plants.

Two hypotheses were considered in explaining the difference in toxicity of small and large initial populations. The first assumes that the mealy bugs from a single colony are equally toxic and that various quantities of toxic secretion are necessary, dependent on the general toxic level of the colony and the susceptibility of individual plants. The second assumes that the capacity to produce wilt is limited to certain individuals in the colony and that the development of wilt is governed by the incidence of toxic individuals. Because of the sedentary habit of the insect, it was considered unlikely that great variation in toxicity would occur among individuals of a single colony. A carefully planned field experiment in which a large number of plants were infested with mealy bugs, varying in number from 1 to 40 per plant, favored the first hypothesis and led Carter (1935) to conclude that "the most susceptible plant required a toxic dose greater than that provided by the feeding of one mealy bug," although in later experiments occasional plants wilted following infestation by single mealy bugs (Carter 1937). In general, the percentage of plants wilting increased with the number of mealy bugs, but the increase was not directly proportional. There appeared to be a point on the dosage scale that induced a disproportionate increase in wilt, dosages beyond this point resulting in only small increases in the number of wilted plants.

Green Spotting of Pineapple and the Pineapple Mealy Bug.—When Illingworth (1931) described the mealy-bug wilt of pineapple, he mentioned a characteristic green spot associated with the feeding punctures of the mealy bug [*Pseudococcus brevipes* (Ckl.)] and interpreted it as a symptom of wilt. Carter, however, in later investigations (1933b) showed that some of the mealy bugs did not cause green spotting even though they readily caused wilt. He concluded, therefore, that green spotting is a disease separate and distinct from pineapple wilt although both diseases are caused by the toxic secretions of the same insect.

Mealy-bug colonies on the pineapple are found most commonly feeding on the white or light-green tissues at the base of the leaves.

If the colonies are very large, they may extend up the leaf for some distance on the greener tissues. The green spots therefore are first seen on the white tissue as faint yellow-green homogeneous spots. As the leaf grows and becomes darker green, the spots maintain a darker color than the surrounding tissue. Often there is a concentric zone of light green around the darker center. Some of the spots may appear as raised welts, especially in older and partly shriveled tissues. Occasionally the center of

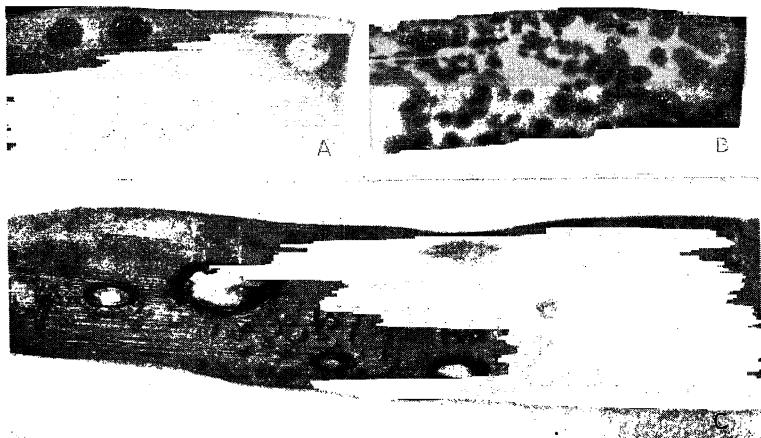


FIG. 59.—Green spotting of pineapple. Photographs showing several of the various types of spot. A, large green spots with dark centers; B, green spots in which color has persisted while surrounding tissue has become extremely chlorotic; C, a leaf with three distinct kinds of spot: raised, weltlike spots in median area, nonelevated green spots near margin of leaf and several large spots of secondary necrosis. (After Carter.)

the spots may become chlorotic and eventually necrotic. The spots vary in size from 1 to 10 millimeters in diameter, averaging 3 to 5 millimeters (Fig. 59).

These spots are strikingly different from the feeding injuries made by a strain of mealy bugs that does not cause the green spots. These are usually visible as small, irregular-shaped, chlorotic spots with some rugosity of the leaf surface if the punctures are numerous. The spots usually are the result of a number of mealy bugs feeding for a prolonged period in one

place. Histological examination of the typical green spots reveals an increase in size and number of chloroplasts with no thickening of the cell walls. The protoplasm appears normal but contains flat polygonal bodies that stain heavily with Lugol's solution. Often the green tissues will appear as an island of green in chlorotic tissue. In the chlorotic spots formed by the feeding punctures of nongreen-spotting mealy bugs, the protoplasts are degenerate, and the cell walls are thickened. The area of injury is more localized and does not extend far beyond the actual area fed upon.

It was observed that those mealy bugs which regularly cause the green spots are a darker gray color than the pinkish gray ones which do not cause the green spots. These darker individuals appear to move about more frequently as if the green spots make the tissue unsuitable for further feeding. Further examination of the insects showed that the dark-gray color of the green-spotting mealy bugs is caused by the presence of a dark-colored mycetome, the mycetomes of the "normal" mealy bugs being white. A microscopic study of the mycetome showed that it contains two different kinds of microsymbiotes. A yeast-like microsymbiote is always present in the mycetome of both types of mealy bug, and a bacterium-like, rod-shaped symbiote is found only in the dark-gray green-spotting mealy bugs. Further studies proved that the bacterium-like symbiote was pleomorphic and that by passing through a transitional stage it was transformed into a coccus form in the pink non-spotting mealy bugs (Fig. 60.)

It was further observed that mealy bugs taken directly from a grass host (*Panicum barbinode* Trin.) contained the symbiote in the coccus form and failed to cause the green spots. In a series of feeding experiments, in which green-spotting mealy bugs were removed from pineapple and fed on *Panicum* grass for two or more generations and then returned to pineapple, the green-spotting capacity was lost. Parallel with this loss, the bacterium-like symbiote changed from the rod form to the coccus form. Continued cultivation on pineapple did not restore the green-spotting property of the mealy bugs or the rod form of the microsymbiote, indicating that the change was not reversible. From these and other experiments Carter concludes that the green spotting is caused by a phytotoxic secretion of the mealy

bug that is conditioned by the presence of the microsymbiote in the rod-shaped form.

Although the parallelism between the presence of the microsymbiote in the rod form is striking, it does not necessarily prove that there is a direct relationship between the two. There seems to be a possibility that the same factors that influence the toxicity of the insect's secretion also influence the mor-

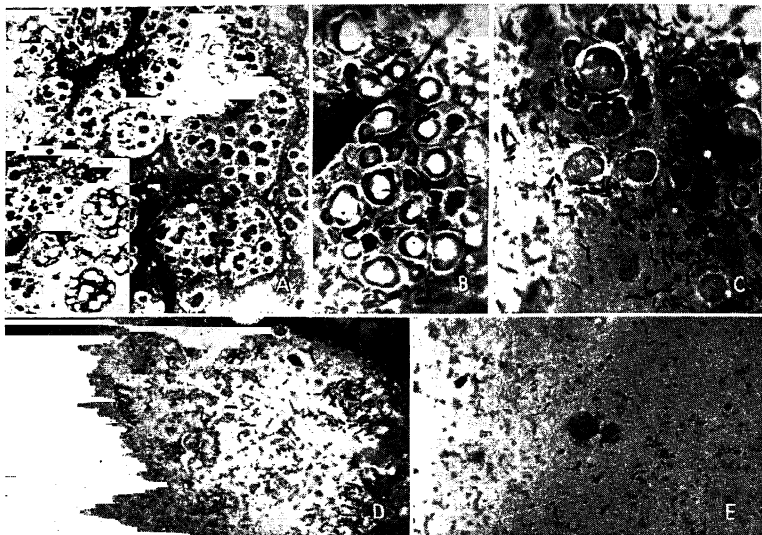


FIG. 60.—Photomicrographs showing the microsymbiotes of *Pseudococcus brevipes*. A, mycetocytes containing the common form of symbiote; B, a preparation showing the rod-shaped form interspersed with vacuolated cells of the common form; C, rod-shaped symbiote from green-spotting mealy bug; D, intermediate forms of rod-shaped symbiote; E, coccus form of symbiote. (After Carter.)

phology of the microsymbiote. This would not necessarily imply a causal relationship. The probability that the morphology of the microsymbiote conditioned the toxicity of the secretions would be greater if it were known that the microsymbiotes had any direct connection with the oral secretions of the insect. There is as yet no proof that this is true. The mycetome is found in close apposition to the mid-gut, but there is no direct connection. Neither has there been shown any connection of the mycetome with the salivary glands. Carter

(1936b) states, "The invariable presence of the rod-shaped form in green-spotting mealy bugs, the evidence of a transitional form, the complete absence of the rod, and the invariable presence of the coccus rod in nongreen-spotting mealy bugs are considered proof of the relationship between the symbiotes and the oral secretions of the insects." These facts may show strong indications of a relationship, but they could scarcely be considered as final proof of so complex a physiological relationship. Further evidence will be necessary before the causal relationship of the pleomorphic changes in the microsymbiote and the toxicity of the secretions of the insect can be accepted as a proved fact.

Ito (1938) has studied the life histories of the pink and the gray strain of this insect. It was found that the pink form reproduced parthenogenetically, producing only females, while the gray form reproduced sexually, producing both males and females. This discovery may prove of some importance in future studies, although its significance is not obvious at present. In other aspects of their life histories, there was very little difference.

Psyllid Yellows of the Potato. In 1928, a new and destructive disease of potatoes and tomatoes was reported by Richards from the western part of the United States. Richards and Blood (1933) have shown that the disease is definitely associated with the feeding processes of the nymphs of the tomato psyllid (*Paratrioza cockerelli* Sulc.). Unlike hopperburn and the russeting of apples by the capsid bugs, psyllid yellows is a systemic disease. Although the insects may feed on only a few of the leaves, the growth of the entire plant is affected to a striking degree.

The symptoms vary somewhat with the number of insects on the plant and the length of the feeding period. The injury is greater when the insects are abundant, but the degree of injury is not always in proportion to the number of insects present. The feeding of 10 to 30 nymphs is necessary for the full expression of the disease. The intensity of the light also has a marked effect on the disease, the symptoms being more pronounced in bright sunlight.

There is a marginal yellowing and an upward rolling of the basal parts of the leaflets (Fig. 61). The terminal leaves of the pigmented varieties assume a red or purple cast. In advanced stages, the older leaves become necrotic. The entire plant is

stunted by failure of the stems to elongate, the nodes swell, and the axillary buds may develop into aerial tubers or short shoots with swollen bases and small distorted leaves. Tuberization is suppressed in younger plants, and the stolons of older plants may develop secondary aerial shoots. The physiology



FIG. 61.—A leaf from a potato plant affected with psyllid yellows showing the curling at the bases of the leaflets. (After Richards and Blood.)

of the entire plant is affected strikingly, and the translocation of photosynthetic products is decidedly abnormal (Fig. 62).

There has been some difference of opinion as to the nature of the disease. Binkley (1929) reported evidence which led him to conclude that the disease was caused by a virus transmitted by the psyllid nymphs, and Shapovalov (1929) observed what he thought was tuber transmission of the disease in potatoes.

The most thorough and complete investigation of the disease to date is that of Richards and Blood (1933). These authors were unable to demonstrate tuber transmission and could not prove the virus nature of the disease, although the possibility of its being caused by a virus was recognized. They showed that when the insects were removed from affected plants there was a cessation of the development of symptoms and a uniform tend-



FIG. 62.—A potato plant affected with psyllid yellows. (After Richards and Blood.)

ency to recover. The disease is produced only by the nymphs, the adults being nontoxiniferous. The ability to produce the disease is inherited by the insect without regard to its having fed on a diseased plant. Richards and Blood did not reach a final conclusion as to the nature of the disease, but the evidence presented strongly favors the view that the psyllid is a toxicogenic insect and that the disease is caused by a toxic substance injected into the plant by the nymph in the act of feeding.

Smith (1937) has classified psyllid yellows as a virus disease caused by *Solanum virus 18*. This does not appear to be justified on the basis of the available evidence.

Psyllids in general have well-developed mycetomes that harbor microsymbiotes (see Chap. III). In the light of Carter's work on the relation of microsymbiotes of mealy bugs to the green-spotting disease of pineapple, a study of the microsymbiotes of this insect seems desirable. Such a study of the tomato psyllid offers a good opportunity for testing the hypothesis that microsymbiotes are in some way concerned in the production of toxic substances secreted by the toxicogenic insects.

Eyer and Crawford (1933) have studied the feeding habits of the tomato psyllid and the pathological histology of the disease. The nymphs feed primarily on the leaves, usually on the lower surface, seeking out the vascular bundles. Their setae terminate most frequently in the phloem tissues or in the border parenchyma surrounding the phloem. There is no extensive injury or plugging of the xylem, although phloem cells are often killed. A definite accumulation of starch is found in the parenchyma tissue, reflecting the inhibited translocation of photosynthetic materials. A more detailed study of the abnormal physiology of carbohydrate translocation has been reported by Eyer (1937).

The life history of the tomato psyllid has been described by Knowlton and Jones (1934) and by Daniels (1937). The insect feeds on both wild and cultivated species of solanaceous plants. It hibernates in the adult stage in warm, dry locations and in early spring feeds on clumps of wild perennial species of ground cherries (*Physalis lanciolata* Michx., *P. longifolia* Nutt., and *Quincula lobata* Raf.). One or two generations may be spent on these weeds before migration to the potato fields begins. The small, spindle-shaped eggs are deposited most abundantly on young apical leaves on either the lower or the upper surface and are attached and supported by a short stipe (Fig. 63). From 5 to 150 eggs per day are deposited by a single female during an oviposition period varying from a few days to several months. The insects are favored by warm, dry weather, and in favorable seasons there may be 8 to 10 generations. The eggs hatch in about five or six days and the nymphs crawl down the stipe and begin feeding on the plant tissues, passing through five instars. About 15 days are required for the completion of nymphal

development. There is usually a high mortality of nymphs at the first ecdysis. The adults are winged and are quite active when the plants are disturbed.

List (1939) has reported on the relation of temperature to the development of the psyllid and to its prevalence. He concludes that it thrives best at about 80 degrees and is retarded in its development at higher temperatures. A temperature of 95 degrees for only 2 or 3 hours per day permits little, if any, increase in numbers. Its prevalence was correlated with

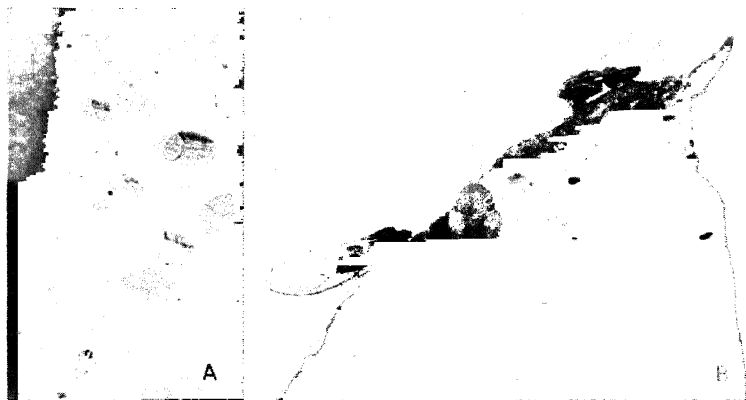


FIG. 63.—The tomato psyllid. A, section of a leaf enlarged to show eggs and nymphs (approx. 10 \times); B, two adults, a young and old nymph, and several eggs on a potato leaf (approx. 12 \times). (Courtesy of Montana Agricultural Experiment Station.)

temperatures in extensive field observations of a period of more than twenty years.

The relation of temperature to migration and hibernation of the psyllid is discussed by List, who expresses the opinion that the psyllid hibernates in some breeding ground distant from the region of high summer infestations. It is thought possible that with the coming of hot weather in the southern breeding areas the psyllids migrate and are brought into the farming regions by the same air currents that bring in the beet leaf hoppers. This hypothesis is supported by the observations of Romney (1939), who has reported that the psyllids breed abundantly in early spring on *Lycium andersonii* Gray and *L. macrodon* Gray

in the semidesert areas of southern Arizona. By the middle of June, they have all migrated and are not to be found again until an influx occurs in late October or early November. There is obvious need for more extensive studies of the breeding and migration habits of this insect in relation to the prevalence of psyllid yellows.

Successful control of psyllid yellows has followed the control of the insects by spraying with lime-sulfur (List 1934, Daniels 1934, 1937). Liquid lime-sulfur spray (1 gallon, 32 degrees Baumé, to 40 gallons of water) applied with a power sprayer at 200 to 300 pounds pressure is recommended. Two applications early in the growing season are required.

Anasa Wilt of Cucurbits.—A wilt of squash, pumpkin, and other cucurbits caused by the squash bug (*Anasa tristis* De G.) was described in 1931 by Robinson and Richards. It was said to be responsible for the complete abandonment of squash and pumpkin growing in many parts of Utah. The disease, which resembles bacterial wilt in many respects, is caused entirely by the feeding activity of the bug, there being no parasitic micro-organism involved. Under experimental conditions, wilting occurred in 24 hours to 16 days, depending upon the number of insects, age of plant, and other factors.

If the insects were removed before wilting had progressed too far, the plants recovered and the new growth was normal. Only the parts above the point of insect feeding were affected. Because of the rapid rate of development and the small number of insects required to cause wilting, it was concluded that the disease is caused by the injection of a toxic substance into the plant by the insect during the feeding process.

Insect Galls.—Galls caused by insects constitute a pathological condition that should be classified with those diseases caused by toxicogenic insects. It is agreed by nearly all those who have studied the subject that the galls develop in response to a stimulating substance introduced into the plant by the insects. In practically all insect galls, the stimulus to abnormal overgrowth arises from the young insects that develop inside the plant tissues. There is some evidence that the females of certain gall-producing insects secrete an irritant that is injected into the plant at the time of oviposition. An enlargement of the tissue takes place before the egg has hatched, probably as a

result of the injected substance, but the presence of the developing larva is necessary for the complete formation of the typical gall.

Cecidiology, or the study of galls, is an extensive subject and has been investigated from many viewpoints. The number of insect species that cause galls is very large. However, insect galls do not cause a great amount of injury to cultivated plants and consequently have received relatively little attention from the plant pathologists. Comprehensive treatments of the subject, chiefly from the academic viewpoint, have been written by Cook (1902, 1903, 1904), Connold (1902), Cosens (1912), Felt (1917), Küster (1925), Ross (1932), and others. It will be necessary here to give only a brief outline of the essential known facts about insect galls.

A gall, or cecidium, is usually defined as an abnormal plant overgrowth caused by a parasitic plant or animal. If caused by a plant stimulus, the gall is known as a *phytocecidium*; if caused by an animal stimulus, it is a *zoocecidium*. The majority of plant galls are zoocecidia and are caused principally by insects and by mites (*Acarina*), especially by species of *Acarus*, *Eriophyes*, and *Phyllocoptes*. The gall-forming insects are found, for the most part, in the Hymenoptera, Diptera, and Homoptera. A few minor galls are caused by species of Coleoptera and Lepidoptera. Most of the gall-producing Hymenoptera are in the family Cynipidae, although a few representatives of other families also are gall formers. According to Folsom and Wardle (1934), approximately 86 per cent of the cynipid galls are found on species of *Quercus*, about 7 per cent on species of *Rosa*, two per cent on the Compositae, and the remainder on other species of plants.

The Diptera include the large family Cecidomyiidae, or gall midges. The North American gall midges and their galls have been studied extensively by Felt (1913 to 1925), who has described more than four hundred species forming galls on roots, stems, leaves, fruits, and buds of nearly two hundred genera of plants. In addition to the gall midges, a few Diptera of the family Trypanidae (Trypetidae), Chloropidae (Oscinidae), and Agromyzidae also produce galls.

The gall-producing Homoptera are confined mostly to the Aphididae and Psyllidae with a few in the Coccidae. The gall

aphids attack a wide variety of plants, and the Psyllidae cause most of their galls on species of hackberry.

Mites belonging to the family Eryiophyidae cause leaf and bud galls. One group of mite galls is characterized by the excessive production of abnormal trichomes. The diseased condition is frequently spoken of as "erinoe," and the galls are often called "felt galls." The trichomes may be free on the leaf surface or enclosed in pouchlike concavities resulting from the mite infestation. Other mites of the family infest buds, causing them to swell but inhibiting normal unfolding, a condition usually known as "big bud."

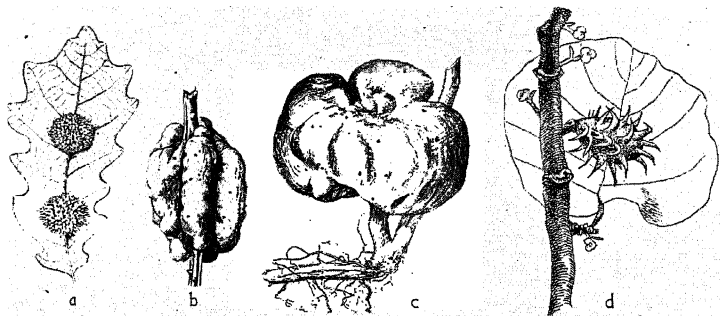


FIG. 64.—Representative types of insect gall. *a*, a leaf gall of oak caused by *Phylloxera prinoidea*; *b*, a stem gall of blackberry caused by *Diastrophus nebulosus*; *c*, a root gall of rose caused by *Rhodites radicum*; *d*, a bud gall of witch-hazel caused by *Hamamelistes spinosus*. (After Felt.)

Insect galls assume a wide variety of sizes and shapes. All gradations from the apparently normal to the decidedly abnormal are found. It is not easy to draw a line distinguishing true galls from deformations not worthy of classification as galls. A definite transition from the simple to the more complex can be observed. The more simple galls may be formed by a mere folding of the leaf lamina. More complex sac galls are formed by invagination of the leaf surface. The more highly developed cambial galls result from insects developing deep in the tissues of the stems, usually after the insertion of the egg at time of oviposition. The form of the gall and its position on the host plant are usually very characteristic of the insect, and the species

can often be identified by the character of the gall it produces. Insect galls are often classified on the basis of the part of the plant on which they characteristically are found. Four classes are usually recognized: (1) leaf galls, (2) stem galls, (3) bud galls, and (4) root galls (Fig. 64).

The physiology and mechanics of gall formation are known only imperfectly. The detailed structure of many galls and the insects causing them are known, but the physiological processes involved in gall production are still obscure. Gall tissues are formed by hypertrophy or hyperplasia or both. Hyperplasia is the most common type of tissue change involved in the formation of insect galls.

In nearly all galls, the growth of the gall is associated with the development of the immature stage of the insect. Certain Homoptera, especially the aphids, may cause pseudogalls at any age by external stimulation. A few instances have been reported where a certain amount of tissue enlargement has followed oviposition without larval development, but such stimuli are not sufficient for the formation of a typical gall.

It is of interest that alternate generations of certain gall insects may produce different kinds of galls. Pergande (1901) has described the life history of *Hamamelistes spinosus* Shimer, showing that different generations are capable of producing different kinds of gall on the alternate hosts, while certain generations of the same species are unable to produce any kind of gall. This phenomenon was discussed by Lutz and Brown (1928), who pointed out that the agamic generation produces a gall quite different from that produced by the sexual generation. These may or may not occur on the same plant but never on the same part of the plant in question. It is generally assumed that the immediate stimulus is brought about by the secretions of the developing larvae, although Cook (1902, 1903, 1904) was inclined to the viewpoint that the mechanical stimulus furnished by the feeding larvae was sufficient to bring about excessive growth of the plant tissues. Adler (1881) and Beijerinck (1888) expressed similar views. Symbiotic microorganisms have been suspected of being involved also in some cases of gall production. Lutz and Brown (1928) found a bacterium constantly associated with the aphid, *Hamamelistes spinosus*, and the bud gall of witch hazel. They suggested that the bacteria furnish

the stimulus to gall formation, but they failed to prove this point experimentally.

Neger (1913) and others have described a number of midge galls in which certain fungi are constantly associated with the insect. Neger concluded that there is a symbiotic association between the fungus and the insect, and because of the similarity of the association to that of the ambrosia beetles he called the galls "ambrosia galls."

Ross (1922, 1934) has studied extensively a number of these fungus-infected galls. He believes, however, that the fungus lives in the gall as a harmless saprophyte until the larvae mature and emerge, when it becomes parasitic on the plant and causes a decay of the gall. It is not known definitely how the fungi first gain access to the gall tissues, but it is believed that they are carried in by the larvae. The species of fungi and the extent of their development in the galls vary widely. Species of *Macrophoma* and many yeastlike fungi have been isolated in pure culture. According to Ross, the fungus is not essential to the development of the gall or of the insect. He also does not consider its association with the insect one of symbiosis and objects to calling them ambrosia galls.

Docters Van Leeuwen (1929, 1939) has described a gall on *Symplocos fasciculata* Zoll. in Java caused by a gall midge (*Asphondylia bursaria* Felt), which he interprets as living in symbiosis with a fungus. When the galls mature in December, the larva within the gall is completely surrounded by a mass of fungus mycelium. The fungus mass is black on the surface but white internally. This internal white portion is completely consumed by the larva before it reaches maturity. The adult insects emerge in February and March and deposit eggs in the stems, petioles, and mid-ribs of the leaves. It was observed in a study of the life history of the insect that each egg is constantly contaminated with spores, but the method of contamination was not determined. As soon as the gall tissues are well differentiated, the fungus grows rapidly and fills the entire chamber except the small space occupied by the larva. The gall could not be produced with either the fungus or the insect independently. All attempts to grow the fungus in artificial culture failed. It is not known definitely, therefore, what influence the fungus has on the development of the gall tissues. However,

Docters Van Leeuwen (1939) agrees with Neger that the relationship between fungus and insect is one of true symbiosis and considers the gall an ambrosia gall.

Malpighi was perhaps the first to suggest that the stimulus to gall formation was of a chemical nature. This conclusion, dating from the seventeenth century, has been reached by many workers of more recent date. Rössig (1904), Triggerson (1914), Magnus (1914), Kostoff and Kendal (1930), Zweigelt (1931), and others have presented evidence to support the view that chemical substances secreted by the young insects are the principal stimuli responsible for the abnormal tissue growth. There is very little known, however, of the nature of the chemical substances or the mechanism of the changes that they bring about in the plant cells.

Cosens (1912) has given consideration to the physiology of gall formation and has concluded:

The larva secretes an enzyme capable of changing starch to sugar which acts on the starchy constituents of the nutritive zone and accelerates the rate of their change to sugar. The material thus prepared supplies nourishment for both the larva and the gall. The protoplasm of the latter is thus rendered unusually active since it receives an abnormal quantity of available food material in a limited area. The hypertrophy and cell proliferation and probably the appearance of vestigial issue or other primary characters are the response of the protoplasm of the host to the additional food supply.

The experimental proof of the nature of the physiological process is very difficult, and all attempts at artificial production of typical galls by the injection of extracts or other chemical substances have failed. Gall production is a very complicated process; and even though simple chemical substances secreted by the insects may be largely responsible for stimulating overgrowth, the presence of the developing larvae appears to be necessary for typical gall production.

The relationship between insects and plant diseases in gall formation has been discussed more recently by Felt (1936), who points out that growth in a plant is probably controlled in part by diffusion from one tissue to another and that any single meristematic cell is capable of forming a part of any plant structure normal for the species. Potentially, any living

cell is capable of meristematic growth, and its behavior is determined by its position in the living tissue. Felt concludes:

The production of plant galls is dependent on stimulation of plant cells in a meristematic or plastic condition. A large proportion of plant galls are produced in buds or in tissues as they develop from buds. The gall producers stimulate cell production in the earlier stages of gall formation. The possibility of these remarkable developments depends on the adaptability of plant cells or groups of plant cells. It can hardly be held that these growths are of material advantage to the plant. They seem to be, on the part of the plant, a more or less blind reaction of cell groups, as it were, to stimuli which, under other conditions, might result in the production of buds and the parts ordinarily developing from them. There is a close analogy between the insect gall and the development of adventitious buds and an actual approach, in methods of growth and formation, to plant seeds or fruits, the gall producer occupying the place of the seed and the surrounding tissues being comparable to the husks or carpels.

The histology of insect galls has been studied extensively. The tissue changes associated with gall production may be either *kataplastic*, in which the gall tissues show little or no differentiation, consisting mostly of large, thin-walled parenchyma cells, or *prosoplastic*, in which the tissues are well differentiated, often being more complex than normal tissues. The majority of insect galls are prosoplastic. Kataplastic galls are more commonly produced by fungi and bacteria. In well-formed, prosoplastic insect galls, three distinct kinds of tissue may be recognized: protective tissue, mechanic tissue, and nutritive tissue. The outer tissues made up of epidermal cells or tissues derived from them are definitely protective in nature. The protective tissues may be little changed from normal or greatly modified by the excessive production of such additional protective structures as trichomes. Stomata and lenticels are present, but the stomata do not close so readily as those on normal tissue. The mechanic tissue is composed of sclerotic cells (stone cells) or heavily lignified fibers that surround the developing larva and protect it from outside pressure. The nutritive tissue is that immediately surrounding the young insect and the one from which the larva derives its nourishment. It usually consists of thin-walled cells with dense protoplasmic contents.

In addition to these three tissue types, some galls have the so-called "aeration tissues" composed of loosely arranged cells with large intercellular spaces. It has been assumed that such tissues provide for easy access of oxygen to the developing larva. Vascular tissue is present in amounts adequate for the needs of the gall and is normal in every respect. An effective vascular system appears to be necessary for the excessive growth required for the development of a prosoplastic gall.

Trichomes, glands, and other epidermal structures are often produced in excessive quantities by galls, and many such structures may appear to be new and atypical for the plant species; but, according to Cosens (1912), the galls cannot produce any fundamentally new tissues. He states that a careful search of the normal plant has always revealed the structure in question, although it usually occurs in much smaller numbers.

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CHAPTER VI

INSECTS AND BACTERIAL DISEASES

Approximately two hundred species of bacteria have been proved pathogenic to plants. Some of these are extremely destructive, while others are of minor economic importance. Insects play a major role in the epiphytology of several of the most destructive bacterial diseases and influence to some extent the development of many others. At least one important bacterial disease, cucurbit wilt, is entirely dependent upon insects for its spread and development.

The plant pathogenic bacteria are small, rod-shaped, unicellular organisms averaging less than 1 micron in width and less than 2 microns in length. They are unable to penetrate the uninjured cuticle of plants and therefore must gain entrance through natural openings or through injuries. The wounds made by insects are utilized for this purpose by many bacterial plant pathogens. Bacteria and bacterial diseases have many other characteristics that make them well suited to insect transmission.

The literature is replete with references to possible relationships between insects and bacterial diseases. When the method of dissemination of a bacterial pathogen is not clearly understood, the statement is very frequently made that it is "probably spread by insects." Such statements usually are not supported by experimental evidence, and often no specific insect is named. In other cases, specific insects may be mentioned, but conclusive evidence of their incrimination is often lacking. Not all these references are included here. In the following pages, some of the better known relationships are discussed, and an attempt is made to weigh the evidence and to evaluate the significance of the insects in the development of the diseases. For more complete information on bacterial diseases of plants, the reader is referred to the works of Smith (1905, 1911, 1914), Elliott (1930), and Stapp (1929) and the literature cited by them.

Fire Blight of Orchard Fruits.—Fire blight is a plant disease of unique historical interest. In 1881, Thomas J. Burrill proved that the disease was caused by bacteria, demonstrating for the first time that bacteria could cause a disease of plants. About ten years later, in 1891, Waite proved that bees and wasps were active vectors of the disease. This constituted the first experimental proof that insects were of importance as vectors of any plant disease.

Fire blight is chiefly a disease of pears and apples although it is not strictly confined to these crops. Other orchard fruits, as



FIG. 65.—Pear blossoms killed by fire blight. (After Rosen.)

well as many ornamental plants, are often affected, although less severely than pears and apples. The known host range is wide, including, according to Thomas and Thomas (1931) and Thomas and Ark (1934a), 90 or more species, mostly in the family Rosaceae. Fire blight is a major disease of apples and pears, and it is a limiting factor in pear production in the Mississippi Valley region of the United States. The disease was first reported in Eastern North America where it has been known since 1794. It is known to occur also in Japan, Italy, and New Zealand.

The disease affects primarily the blossoms (Fig. 65) and young twigs (Fig. 66) of apple and pear trees, but infection may occur

also in larger branches, causing destructive cankers. The cankers often completely girdle and kill the large branches. When the trunk of the tree is affected, the condition is called "collar blight." Pear trees and trees of the most susceptible varieties of apple may be killed outright by fire blight alone. On the more



FIG. 66.—Twig blight of pear. Although infection may occur through stomata or mechanical injury, twig blight most commonly follows inoculation by sucking insects. (After Rosen.)

resistant varieties, fire blight itself may be less destructive, but it is frequently followed by black rot and other fungus diseases that eventually destroy the tree.

Fire blight is caused by bacteria [*Erwinia amylovora* (Burrill) Winslow *et al.*] that are favored by warm, humid weather and are most virulent on succulent, rapidly growing tissue. Our knowledge of the epiphytology of fire blight is incomplete, and

there is a wide diversity of opinion among those who have investigated the subject. Early investigators recognized that the pathogen could live over winter in the cankers on the larger branches, and it was generally assumed that these "holdover" cankers constituted the chief, if not the only, means of survival. However, in recent years several other possibilities have been suggested. The sources of primary inoculum in the spring and the relative importance of the different means of dissemination of the pathogen constitute the crux of the fire-blight problem.

As early as 1884, Forbes observed fire-blight lesions associated with the feeding activities of the tarnished plant bug (*Lygus pratensis* L.) and expressed the opinion that this insect was acting as a vector of the disease. Forbes, however, presented only observational evidence for his conclusions. The accuracy of his observations was confirmed by experimental evidence presented by Stewart (1913*a*, 1913*b*) nearly thirty years later.

Waite (1891) proved experimentally that bees and wasps become contaminated with the fire-blight pathogen when visiting blighted pear blossoms and that these insects commonly spread the infection to other blossoms. The most complete account of Waite's work is given by Smith (1911, Vol. II, p. 55) who states:

Mr. Waite again produced blossom-blight on certain clusters of pear-blossoms by infecting the floral nectaries and by allowing the bees to have free access to these blossoms. He succeeded through their agency in transmitting blight to other flower-clusters on the same tree. One of these experiments took place on the grounds of the United States Department of Agriculture, an isolated tree previously free from blight being used for this purpose. Bees were observed to visit the infected flowers and, subsequently, flowers on other clusters, which flowers afterwards blighted. Some of these bees were caught, their mouth parts excised, and cultures made therefrom by means of poured-plates in Petri dishes. Colonies obtained in this way closely resembled the pear-blight organism, and inoculations therefrom produced the disease in sound pear-shoots, thus demonstrating beyond dispute the actual presence of the pear-blight organism on the mouth parts of the suspected bees.

Everybody connected with the plant pathological work of the United States Department of Agriculture at that time had knowledge of these results. The writer, among others, saw all of the experiments described and knows that they were well done and that the above brief outline can be accepted as an accurate statement of what actually took place.

Being familiar with the holdover cankers and the bacterial exudate frequently associated with them in early spring (Fig. 67), Waite concluded that bees, wasps, and other insects visit the exudate, become contaminated with the bacteria, and disseminate them to the blossoms. But this conclusion was not based on experimental evidence. All his experiments were concerned with the spread of secondary inoculum. Conclusions



FIG. 67.—A holdover canker of fire blight on apple showing the bacterial ooze, from which much blossom infection originates. Flies and ants feed upon the ooze and transfer the bacteria to blossoms where they infect. Secondary spread is accomplished largely through the activities of the honeybee. Rain water is also important in both primary and secondary spread of blight. (After Whetzel.)

concerning primary infection were based on observation and assumption only, and later work indicates that bees rarely visit the exudate. Writers, in reporting Waite's work, often have accepted the assumed role of bees in primary infection as a proved fact, and this explanation was accepted generally for many years. Recently several workers have questioned the importance of holdover cankers in primary infection and offered alternative theories based on more or less experimental evidence. On the other hand, Thomas and Ark (1934a) have reported direct experimental proof that flies and ants carry blight bac-

teria from holdover cankers to blossoms and initiate primary infection.

Gossard and Walton (1922) were the first to emphasize the importance of rain drip and wind-blown rain as a means of dissemination of secondary inoculum, but they did not appreciate its possible significance in primary infection. These workers, by means of cage experiments, proved conclusively the effectiveness of rain in disseminating the bacteria from blossom to blossom. They verified the earlier work of Waite showing that bees are vectors, but they considered rain to be the most important agent of dissemination after a few centers of primary infection had been established by insects. The proof of the importance of rain water in primary infection has been demonstrated by Brooks (1926) and confirmed by Miller (1929), Tullis (1929), and others who maintain that the greater part of primary infection results from inoculum from "holdover" cankers splashed or blown to blossoms in rain water. Thomas and Ark (1934b), on the other hand, claim that rain water is of little importance in actual dissemination but that it has an indirect effect in providing favorable conditions for infection after dissemination by insects has taken place. They point out that in dry weather the nectar in blossoms is too concentrated for growth of the bacteria and that the more dilute nectar occurring in rainy weather is more conducive to infection. Another indirect effect of rainfall is pointed out by Shaw (1935) who has shown that apple and pear shoots are more susceptible in wet weather because of the increased intercellular humidity, which is favorable for infection and development of the disease. Pierstorff (1931) concluded, "Meteoric water did not appear to spread the blight bacteria from blossom to blossom." Similar conclusions were reached by Parker (1936).

Gossard and Walton (1922) recognized the wide host range of the disease and proposed a new theory to account for primary infection without the aid of holdover cankers. This is the theory that the blight may be spread from the far South by insects and rain from blossom to blossom in a progressive wave northward. After such a wave had started in the South, it would spread progressively northward with the seasonal advance, infecting all orchards in its path, making the presence of holdover cankers relatively unimportant. The theory obviously

is not easily subjected to experimental proof and is not generally accepted.

Because of their feeding habits, bees are concerned primarily, if not solely, with blossom infection and do not account for the infection of shoots and twigs that do not bear blossoms. Jones (1909) and Whetzel and Stewart (1909) were the first to offer an explanation for twig and shoot infection. These workers observed the frequent association of aphids with blighted twigs and concluded that they were the vectors, but no experimental evidence was presented.

A few years later, Merrill (1915) reported a similar correlation between aphid infestation and fire-blight infection. Stewart (1913a, 1913b) presented the first experimental proof of aphid transmission of fire blight and confirmed the early observations of Forbes that the tarnished plant bug is a vector. Later, Stewart and Leonard (1915, 1916) presented evidence that several other sucking insects could serve as vectors. They concluded that the mode of transmission was purely mechanical and that any sucking insect feeding on a susceptible host was a potential vector. Workers in general agree with this conclusion; but some, including Tullis (1929) and Miller (1929), maintain that sucking insects play a very unimportant part in the epiphytology of fire blight.

Much of the earlier work led to the conclusion that, with the exception of blossom infection, the bacteria could enter only through wounds. Since the most frequently occurring wounds on young shoots are those made by insects, it was only natural to attribute twig blight to this method of infection. However, in 1915 Heald showed that the infection of leaves through natural openings (stomata and hydathodes) was common in the state of Washington and suggested that this might explain many infections which have been attributed to insect inoculation. Infection of young leaves in the absence of wounds was verified by Tullis (1929), Miller (1929), and Rosen (1933).

Infections of the larger branches and trunks usually are the result of invasion directly from infected small shoots, but Jones in 1909 demonstrated that such infection often followed the attack of the fruit-tree bark beetle (*Scolytus rugulosus* Ratz). Two years later (1911), he presented definite experimental proof that these insects were important vectors of the disease in

young pear orchards. Orton and Adams (1915) observed that 90 per cent of the cases of the "collar blight" type of fire-blight infection on apples in Pennsylvania were associated with infestations of the bark beetle and considered this insect as a possible vector although their conclusions were based largely on observational evidence.

Gossard (1916) appears to have been the first to investigate the role of the beehive in the epiphytology of fire blight. He found that the fire-blight bacteria could survive for at least 72 hours in honey taken from the hive and concluded that the hive could serve as a distributing center for the fire-blight pathogen during the blossoming period. Later Gossard and Walton (1922) demonstrated the presence of the pathogen on the mouth parts of bees entering and leaving the hives. They also successfully inoculated apple twigs with honey taken from the hives at the same time. Thomas in 1930 showed that the pathogen could survive in the hive on the comb and frames for 55 days and pointed out the danger of translocating hives from one orchard to another.

These experiments incriminated the hive as a factor in secondary inoculation, but there was no conclusive evidence that the pathogen could survive the winter in the hive. The latter possibility was seriously investigated first by Rosen who, in 1930, reported that he had "successfully isolated the fire-blight pathogen from beehive material gathered throughout the summer, winter, and early spring and from the bees themselves obtained from the hives in the early spring prior to the development of blight." Rosen (1933) obtained 21 successful infections out of 3,534 inoculations into young pear twigs with inoculum obtained from the interior of beehives. He pointed out that, in Arkansas, fresh infectious exudate was absent prior to the appearance of blossom blight, which was invariably the first blight to appear, and that blossom blight was abundant in years when there was no rainfall during the blossoming period. These facts led him to consider the hive as a significant source of primary inoculum.

Rosen's conclusions are questioned by Pierstorff and Lamb (1934), who were unable to produce blossom infection by placing hives artificially infested with the pathogen under apple trees, all enclosed within cheesecloth cages. Thomas and Ark (1934a) in California were unable to confirm Rosen's claims that the

pathogen could survive the winter in the hive. They claim that there is not enough evidence to justify the conclusion that bacteria taken into the hive by bees are sources of either primary or secondary inoculum. These workers conclude that, in California, primary infection originates from bacteria carried from holdover cankers to blossoms by ants, aphids, beetles, codling moths, flies, wasps, and yellow jackets (Fig. 68). Parker (1936) also concluded that the pathogen probably could not survive the winter in the beehive and attributed primary infection to inoculum transferred by ants and flies to blossoms from holdover cankers.

Further objections to Rosen's theory were raised by Hildebrand and Phillips (1936), who have made the most thorough

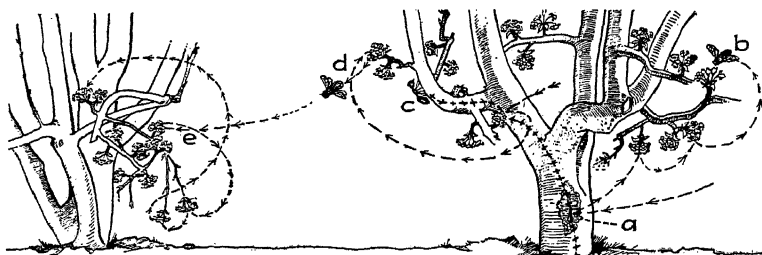


FIG. 68.—A diagrammatic sketch showing the relation of insects to the spread and development of fire blight. *a*, oozing cankers from which flies (*b*) and ants (*c*) transport bacteria to blossoms and initiate primary infection; *d*, bees and other insects that transport bacteria from blossom to blossom, inducing secondary infection. (After Thomas and Ark.)

study yet reported of the relation of the bee and the beehive to the epiphytology of fire blight. They were unable to recover the pathogen from honey, comb, or bees after 3 days, when the pathogen was introduced by feeding the bees on a 60 per cent sucrose solution heavily charged with the bacteria. The longest period of survival in the hive that they could demonstrate was 13 days in pollen and 12 days in frame scrapings. After pointing out that none of the individual worker bees active during an epidemic of blossom blight would be likely to survive until the following spring, they attempted to isolate the pathogen from the eggs, larvae, pupae, and the adult bees in colonies fed on sugar solutions containing the bacteria, but were unsuccessful. It was proved that the bee was important in disseminating the

inoculum from flower to flower and, provided that the bees were feeding on contaminated food, from hive to flowers. It was concluded, however, that the pathogen was incapable of overwintering in the beehive, on the comb, in the honey, or in association with the bee, and that the hive was not a source of primary inoculum in the spring of the year.

This brief review reveals a wide diversity of both opinion and evidence concerning the importance of insects in the epiphytology of fire blight. It is somewhat ironical that the first association between insect and plant disease to be established should, after 50 years, remain in such an uncertain and unsatisfactory state. There are several reasons for this: The nature of fire blight makes experimentation difficult, the size of the suspects makes caging experiments expensive and greenhouse experiments very unsatisfactory, and the widely different environmental conditions prevailing in the different regions where the investigations have been made may explain some of the divergent results.

Much of the work to date has been done with the assumption that the pathogen does not survive in the soil, but recent evidence presented by Ark (1932), that the pathogen may survive for a considerable length of time in the soil, opens up new possibilities that should be thoroughly studied. Hildebrand (1937) has shown that small numbers, even single cells of *Erwinia amylovora*, are highly infectious when inoculated into nectar of apple flowers. He emphasizes the potential infective power of a very small number of bacteria when disseminated by bees from blossom to blossom. The evidence available is sufficient to convince anyone that insects do play an important part in the spread and development of fire blight, but it is practically impossible to evaluate the relative importance of insects and other factors as well as the relative importance of some of the different species of insects that have been incriminated. Much more investigation of the subject is needed. The investigations should have as their main objective the solution of these specific problems and should not be carried out merely as an incidental part of more inclusive studies. They should be made by investigators thoroughly familiar with the techniques of both entomology and plant pathology and with a knowledge of the various associations of insects with plant diseases. The recognized importance of the bee in the pollination of fruits and the increasing attention being given to the control of

blossom blight in orchard practice (Hildebrand 1937a) make it imperative that the relation of bees to the initiation of fire-blight epiphytotics be definitely and accurately established. The problem must be approached with an open mind. The investigators must be thoroughly familiar with the life history, habits, anatomy, and physiology of bees and must have an adequate conception of the nature of the disease and its cause.

Recent work has placed increasing emphasis on flies as agents of dissemination of primary inoculum from holdover canker to blossoms (Thomas and Ark 1934 and Parker 1936). In view of this fact, the work of Ark and Thomas (1936) is very significant. By controlled laboratory experiments, they have shown that the fire-blight pathogen may live for several days in the intestinal tract of *Drosophila melanogaster*, *Musca domestica*, and *Lucilia seriata*. Eggs of *M. domestica* from contaminated females were externally contaminated with the pathogen. It was shown also that when the larvae of *D. melanogaster* and *M. domestica* were fed on contaminated food the pathogen persisted through the puparia and recontaminated the emerging adult. The habit that these insects have of regurgitating the contents of the crop when feeding would make them especially effective as vectors of the fire-blight pathogen. The survival of the bacteria in the puparia opens up the possibility of the pathogen overwintering in this way and suggests another possible source of primary inoculum. These possibilities need further investigation under orchard conditions.

Soft Rot of Plants and Dipterous Insects.—The relation of dipterous insects to the bacterial soft rot of plants constitutes one of the most interesting associations between insects and plant diseases. The relationship was first discovered in a study of the seed-corn maggot as a factor in the development of the blackleg disease of potatoes (Leach 1925, 1926), and it has since been demonstrated for several other insects and plant diseases (Bonde 1930a, Johnson 1930, Leach 1927).

Potato Blackleg.—Blackleg is one of the major diseases of the potato. It causes an average annual loss of approximately 2 per cent of the crop. In badly affected fields the loss may be 50 per cent or more, and it is not uncommon to find fields in which 10 to 15 per cent of the plants have been killed by the disease. In the majority of the fields, however, the losses are seldom greater than 5 per cent of the crop. In addition to reduced yields, there may

be further losses caused by decay of the tubers in storage or in transit. This is especially true where good storage conditions are not possible.

Blackleg is a bacterial soft rot caused by *Erwinia carotovora* (Jones) Winslow *et al.* Any part of the potato plant may be affected, although it is generally the seed pieces, the base of the stem, and the tubers that are directly infected by the bacteria (Fig. 69). The infected seed pieces are usually decayed throughout before the bacteria spread into the stem or out into the stolons to the newly formed tubers. Seed pieces destroyed by blackleg



FIG. 69.—Potato blackleg. A, a potato plant drooping and beginning to die from blackleg infection compared with a healthy plant of the same age; B, an affected plant pulled up showing the dark-colored soft rot of the base of the stem. The seed piece has been completely decayed.

vary in appearance. In the majority of cases, the pathogenic bacteria are accompanied or closely followed by many saprophytic bacteria and fungi that reduce the seed piece to a slimy, foul-smelling mass in which the larvae of insects, particularly the seed-corn maggot (*Hylemyia cilicrura*), frequently may be found. On the other hand, especially late in the growing season, the seed piece may be affected by a watery translucent decay with no unpleasant odor. In such cases, relatively few saprophytes are present.

When a plant becomes diseased after tubers have been formed, the decay frequently spreads through the stolons and into the new

tubers. In wet and heavy soils, these may be completely destroyed before harvest. In light or very dry soils, usually a small portion of the stem end of the tubers will be decayed, or perhaps only a browning of the vascular bundle may result. The flesh of tubers rotted in the soil as a result of stolon infection may remain nearly as white as normal until cut open and exposed to the air, when it will rapidly turn brown or black. Tubers decayed by a pure culture of the blackleg bacteria do not have an unpleasant odor, but when secondary organisms are present a foul

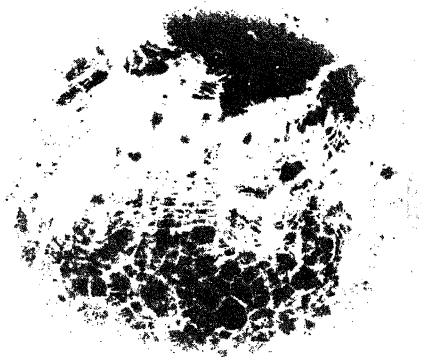


FIG. 70.—A section through a lesion caused by blackleg on a potato tuber showing the wound cork that has successfully checked the decay and walled off the bacteria from the healthy tissues.

odor may result. For a long time, the bacteria causing blackleg of potatoes were thought to be different from those causing soft rot of other vegetables, but more recent work has demonstrated them to be the same (Leach 1930b and Bonde 1939a).

For many years, there was a general belief that the bacteria causing blackleg could not survive in the soil over winter. This belief was based partly on the negative results obtained by Rosenbaum and Ramsey (1918) and Ramsey (1919) and partly on a misunderstanding of the factors influencing infection of potato plants by blackleg. More recently it has been shown that the conclusion based on the earlier work was not justified

and that the bacteria do survive in the soil (Leach 1930a). In fact, the bacteria are present in most cultivated soils and may infect potato seed pieces when conditions are favorable. When potato seed pieces are planted in a soil under conditions favorable for growth, a layer of cork cells is formed over the cut surface and serves as an effective barrier against bacterial infection (Fig. 70). Infection cannot occur unless some condition prevents the formation of this cork layer or enables the bacteria successfully to pass through it.

There are several ways in which the bacteria may succeed in penetrating this protective layer of cork. Numerous experiments have shown that, if the supply of oxygen is low, cork cells cannot be formed. In very wet soils, the air spaces are filled with water; the air being thus forced out, the supply of oxygen is limited. Leach (1930a) has shown that cork formation is greatly inhibited when tubers are planted in very wet soils, but this has no retarding effect on the growth of the bacteria. The bacteria are facultative anaerobes and are able to grow as well in the absence of oxygen as in the presence of abundant oxygen. Under anaerobic conditions, cork cannot be formed by the tubers, but the bacteria may grow rapidly. Thus there are produced conditions favorable for infection of seed pieces by bacteria in the soil. This in all probability accounts for the commonly observed fact that blackleg usually is more prevalent in wet seasons than in dry seasons. It may also explain the greater prevalence of blackleg on heavy, poorly drained soils as compared with lighter well-drained ones.

Insects may aid the bacteria in penetrating the protective cork layer formed by the seed pieces. Investigations by Leach (1926) resulted in the discovery that the seed pieces of plants affected with blackleg were frequently infested by larvae of the seed-corn maggot (*Hylemyia cilicrura* Rond.). Further studies led to the conclusion that this insect is an important agent of dissemination and inoculation of blackleg. Bonde (1930b) has shown that a closely related species (*H. trichodactyla* Rond.) also serves in the same capacity.

The seed-corn maggot is a common pest of the potato and many other plants. Reports of injury on corn, beans, peas, turnips, cabbages, radishes, onions, beets, potatoes, hedge mustard, tomatoes, and several other plants occur in the literature. The insect

overwinters in the soil in the puparial stage. The adult flies emerge in early spring. They may be seen flitting about over the surface of the ground in great numbers during the planting season and appear to be attracted by the freshly turned soil. They are about the size of a housefly, which they resemble in many respects (Fig. 71). Eggs are deposited on, or in, the soil near seed pieces and young potato plants (Fig. 72). The eggs are slightly less

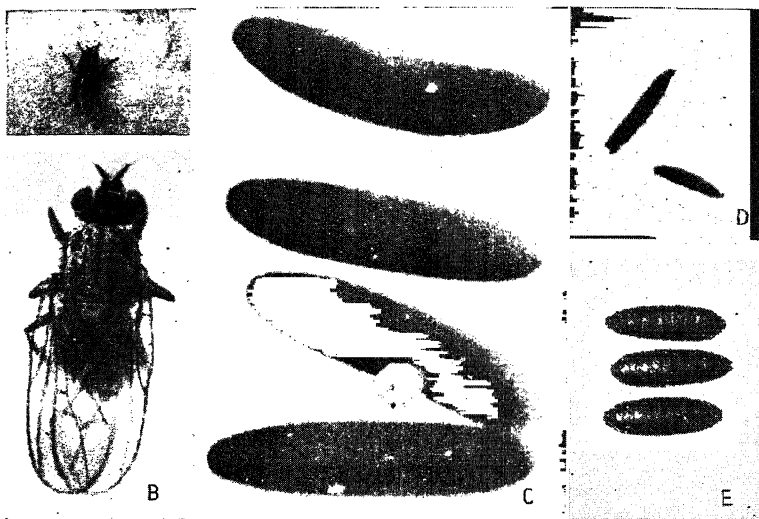


FIG. 71.—The seed-corn maggot (*Hylemyia cilicrura*), the vector of potato blackleg and bacterial soft rot of other plants. A, an adult female fly resting on the surface of a potato tuber (approx. $1\frac{1}{2}\times$); B, a female fly (approx. $9\times$); C, four eggs (approx. $100\times$); D, two larvae (approx. $2\times$); E, three pupae (approx. $3\frac{1}{2}\times$).

than $\frac{1}{25}$ inch in length but may be seen readily with the naked eye. They are slightly curved, white, and distinctly reticulate (Fig. 71C). When freshly deposited, they are covered with a sticky fluid that causes them to adhere to anything with which they come in contact.

The eggs hatch after 2 or 3 days. The maggot, upon emergence, is nearly transparent and only slightly longer than the egg, but it is able to move about rapidly in search of food. The mouth of the maggot is equipped with two black, sharp, clawlike structures with which it is able to penetrate the tissues of the seed

piece. Observations have shown that the maggots during the first 24 hours will crawl about over the cut surface of the seed piece scraping with their mouth parts until the tissues begin to decay. During the next few days, they penetrate deep into the tissues, thoroughly inoculating the seed piece with bacteria. Experiments have demonstrated that the eggs are sometimes contaminated on the surface with the blackleg bacteria when deposited. It has been shown, also, that the bacteria can survive in the soil. The maggot, therefore, may pick up the bacteria

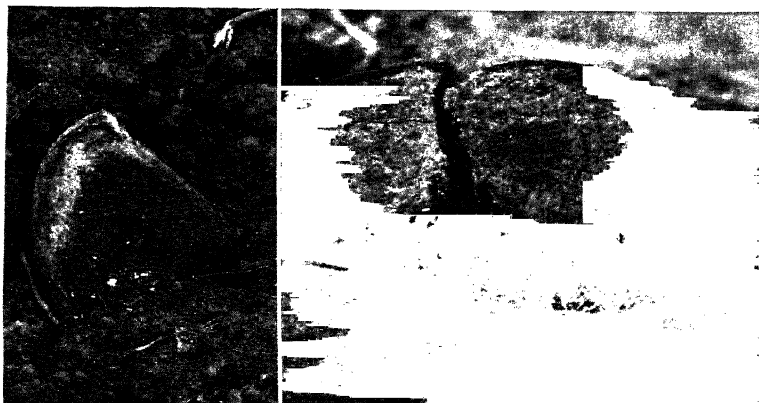


FIG. 72.—Eggs of the seed-corn maggot on, and in the soil near, potato seed pieces. Photographed 3 days after planting.

from the egg shells, from the soil, or perhaps from the surface of contaminated seed pieces. The maggot thus acts as a very effective agent of inoculation. By the continued burrowing of the maggot, any tendency of the tissues to form wound cork is thoroughly overcome.

No effective method of controlling these insects is known, although certain cultural practices may reduce the damage done by them (Leach 1931*a* and Bonde 1930*b*). Bonde (1930*b*, 1939*b*) has shown that the young maggots are attracted to small superficial bacterial lesions on the cut surface of potato seed pieces and that they become established through such lesions more easily than through a sound well-suberized surface. These lesions are usually caused by nonpathogenic or very weakly pathogenic

bacteria that are not capable of causing a general necrosis of the seed pieces or blackleg.

After 2 or 3 weeks' development in the seed pieces, the maggots reach their maximum size of about 7 to 8 mm. They then leave the seed piece, enter the soil, and pupate. In the meantime, the seed piece is usually completely decayed, and the bacteria are invading the stems of the plants, producing typical symptoms of blackleg. Pupation is usually well under way by the time the first aboveground symptoms appear. The puparia are about $\frac{1}{5}$ inch in length and somewhat oval in shape (Fig. 71e). They vary from light brown to dark reddish brown according to age. The duration of the pupal stage varies from 7 to 14 days in summer.

There are usually two broods of the insect on potatoes. The first brood is the more important in its relation to blackleg, for the eggs are deposited near seed pieces or young potato plants. The second brood usually deposits its eggs near or on the stems of plants already affected with blackleg. The maggots of the second brood may frequently be

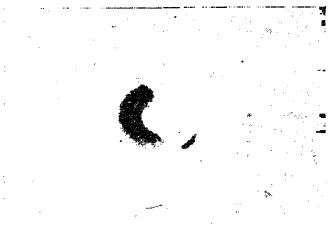


FIG. 73.—Two larvae of the seed-corn maggot showing the effect of bacteria on growth. Both larvae are the same age and were hatched from surface-sterilized eggs. The small one on the right was grown for 12 days on a sterile potato plug. The larger one was grown on a similar potato plug infected with bacteria. $1\frac{1}{2}\times$.

found in the stems of blackleg plants, while the maggots of the first brood are confined chiefly to the seed pieces.

The symbiotic relationships between bacteria and the seed-corn maggot have been studied by Leach (1931b, 1933). It was found that the normal bacterial flora of the intestinal tract of both the larvae and the adult flies is made up of several morphologically and physiologically similar species of bacteria. The most common bacteria are species closely resembling *Pseudomonas fluorescense* Migula and *Ps. nonliquefaciens* Bergey et al. *Erwinia carotovora* is frequently but not always present. These species are regularly passed uninjured through the intestinal tracts of both larvae and flies. It was shown, also, that these bacteria may survive in the puparia and emerge with the adult fly. In the imago, certain species ingested with the food are destroyed and digested while certain others are uninjured. Some of these

actively grow and multiply in the intestinal tract. In neither larva nor imago are the bacteria harbored in special organs such as those described by Petri (1910) and Stammer (1929) in *Dacus oleae* and other Trypetidae.

Nutritional studies (Leach 1926, 1931*b*; Huff 1928) indicate that the bacteria aid the development of the larvae by transforming the plant tissues into a form more readily assimilated. The larvae do not develop normally on sterile potato tubers or on sterilized bean seed but will grow normally if these are contaminated with the bacteria usually associated with the

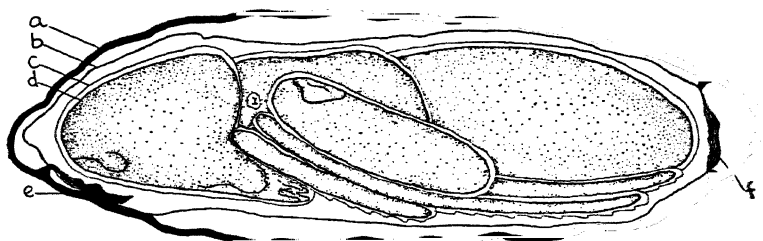


FIG. 74.—A sketch of a puparium of the seed-corn maggot showing the pupa, in perspective, and the various enveloping layers in section. *a*, puparium shell; *b*, prepupal cuticle; *c*, pupal cuticle; *d*, pupa; *e*, extruded lining of fore-intestine of larva; *f*, extruded lining of larval hind-intestine. Approx. 35 X.

insect (Fig. 73). They grow normally on seeds that have been partly decayed by bacteria and then sterilized by heat. The larvae will grow slowly and pupate also on sterile germinating bean seed, indicating that the bacteria, as such, are not essential but that they are needed when the larvae are feeding on non-germinating plant tissues. The specific changes that are essential for the utilization of plant tissues by the larvae are not known. This problem should be investigated further with suitably refined techniques.

Leach (1933) has shown that the bacteria survive in three different locations in the puparium: in the cast-out linings of the fore-intestine, in the cast-out linings of the hind-intestine, and in the lumen of the mid-intestine of the pupae (Fig. 74). The bacteria that survive in the first two localities are those that happen to be in the fore- and hind-intestines at the time of pupation and may be of several different kinds. Those surviving in the mid-intestine are reduced to relatively small

numbers during histolysis but increase rapidly just before the emergence of the fly from the puparium (Figs. 75, 76, and 77). There seems to be a selective action on the bacteria surviving in the mid-intestine that is not operative on those surviving in the cast-off linings of the fore- and hind-intestines. Those surviving in the mid-intestine appear to be of one kind. The specific identity of the bacteria surviving in the mid-intestine has been determined in only a few cases, in all of which a single species of a nonpathogenic bacterium resembling, but not identical with *Ps. fluorescens*, was found.



FIG. 75.—A section through the mid-intestine of a seed-corn maggot larva in early stages of pupation showing the contracted lumen and the cast-off epithelial cells. Practically all the food material has been eliminated, and only a few bacteria remain.

Insect inoculation and direct infection from the soil constitute two sources of infection that may result in severe outbreaks of blackleg without regard to the source of the seed stock. Nevertheless, the disease may be transmitted through infected seed pieces, and this source of infection should not be disregarded. When a plant becomes affected with blackleg late in the season and is not killed until after tubers have formed, it is frequently found that the decay has extended through the stolons and has entered the tubers through the stem end. Such tubers usually are not completely decayed but remain sound with the exception of a slight depression at the stem end and a browning of the

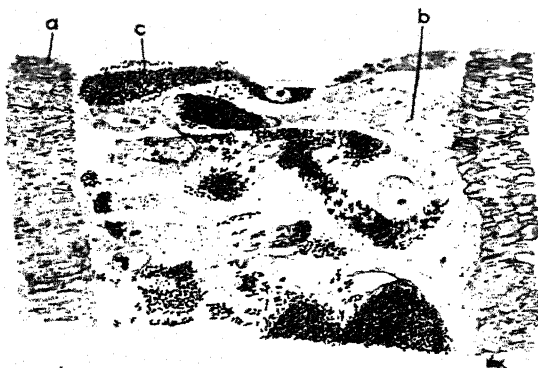


FIG. 76.—A camera-lucida sketch from a stained slide showing the contents of the mid-intestine of a pupa of the seed-corn maggot. *a*, the embryonic cells of the new mid-intestine wall; *b*, the epithelial cells of the old larval mid-intestine being sloughed; *c*, living bacterial cells. Approx. 250 \times .

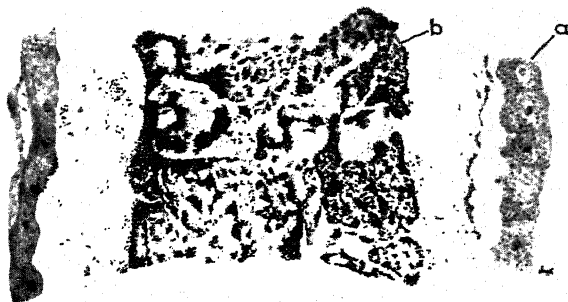


FIG. 77.—A camera-lucida sketch from a stained slide showing a section of the hind-intestine of a freshly emerged imago of the seed-corn maggot. *a*, wall of hind-intestine; *b*, residue of disintegrated epithelial cells; *c*, the bacteria which have survived through metamorphosis and are multiplying on the disintegrated epithelial cells. Approx. 250 \times .

vascular bundles for some distance beyond. Experiments have shown that the bacteria may live over in tubers infected in this way, and such tubers frequently give rise to blackleg plants. Tubers infected in this manner appear to be less effective in walling off the bacteria than those inoculated through the cut surface at planting time. Experiments have shown that only a relatively small percentage (10 to 15 per cent) of such stem-end infected tubers are likely to produce blackleg plants. Therefore, when we consider the number of tubers likely to be infected in this way in a given lot of seed tubers, it appears extremely unlikely that this source of infection could result in more than a very small percentage of the infection frequently found in potato fields.

Soft Rot of Crucifers and the Cabbage Maggot.—Crucifers, in common with many other plants, are subject to bacterial soft rot. The disease is very commonly associated with the attacks of the cabbage maggot (*Hylemyia brassicae* Bouche). Entomologists have frequently mentioned decay of the tissues following maggot injury, but the significance of the association was not known until demonstrated by Johnson (1940) and Bonde (1930a), who proved that the insect was a common vector of the soft-rot bacteria.

Soft rot may affect different species of crucifers in several different ways. One of the most common and most destructive manifestations of the disease is the so-called "stump rot" of cabbage, a decay affecting plants that have started to "head out." The decay originates in the stem but eventually spreads into the base of the head. The first noticeable symptoms usually consist in a wilting of the outer leaves and a cessation of growth. When viewed casually, the head may appear healthy, but further examination will reveal that the interior of the stem is decayed and that the head can be separated readily from the stem, leaving a rotted stump.

This infection practically always originates at the point of maggot attack. This may be below ground, in which case the decay follows the tender tissues of the pith inside the hard vascular ring and may escape observation until the base of the head is thoroughly decayed. In other cases, the decay may originate at the base of the older leaves where the later broods of flies frequently deposit their eggs. Maggot attack does not

always result in stump rot. In many cases, the decay is confined to the succulent tissue of the cortex outside the hard lignified vascular cylinder, and the tissues soon heal.

The symbiotic relations between the bacteria and the insect seem to be essentially the same as those described for the seed-corn maggot. This insect has been mentioned also as a possible vector of *Phytophthora campestri* (Pammel) Bergey *et al.*, the cause of the black rot of cabbage, but adequate proof is lacking.

Heart Rot of Celery.—Bacterial soft rot caused by *Erwinia carotovora* (Jones) Bergey *et al.* is destructive on celery chiefly as a heart rot. Heart rot was first recognized and adequately described by Wormald (1914, 1917). Soft rot may affect any part of the celery plant, but on the older, outer leaves it is of little significance because these are discarded before the celery is packed for market. But when the heart leaves are affected, the central bud is destroyed, the stem elongates, and the plants become unmarketable (Fig. 78). Heart rot should not be confused with blackheart, a nonparasitic disease, which sometimes may be followed by soft rot (Foster 1934).

Weather conditions have a profound influence on the development of heart rot. The influence, however, is contrary to that generally prevailing for the disease on other plants. Destructive outbreaks of celery heart rot occur only in hot dry weather, whereas soft rot of most other plants is favored by rainy weather and high humidity. The explanation of this apparent anomaly is found in the dependence of the disease upon the activity of certain dipterous insects. Two species of leaf-mining Diptera (*Scaptomyza graminum* Fall. and *Elachiptera costata* Leow.) are vectors of the disease (Leach 1927).

These insects deposit their eggs on the leaves of the celery and the young larvae burrow into the leaves. The soft-rot infection starts at the point of entrance of the larvae. The insects normally deposit their eggs in places where the relative humidity is high, and when the eggs hatch the larvae search for a moist place. Observations show that they penetrate the leaf most often where there is free water on the leaf surface. In moist weather, nearly all the eggs are deposited on the older outer leaves. These leaves are more resistant to decay, and if decay does result the outer leaves may be destroyed with no appreciable loss to the grower. In dry weather, the insects

deposit their eggs on the inner heart leaves where the humidity is always high even in the warmest weather. The succulent heart leaves are very susceptible, and the decay develops rapidly until the terminal bud is killed and the plant is a total loss. This disease is prevalent in Great Britain, and the association

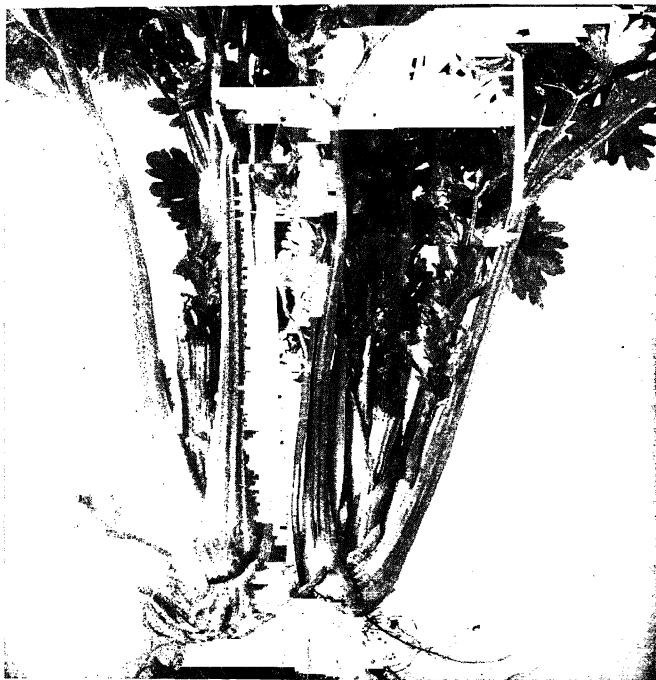


FIG. 78.—A celery plant affected with heart rot caused by *Erwinia carotovora* and transmitted by Dipterous insects, the most important of which are *Scaptomyza graminum* and *Elachiptera costata*.

between *S. graminum* and the soft-rot bacteria has been confirmed by Ogilvie, Mulligan, and Brian (1935). The course of the bacteria in the bodies of these insects has not been studied. It is assumed that the association is somewhat similar to that of the seed-corn maggot, but the facts must await investigation.

Richardson (1938) has shown that the tarnished plant bug (*Lygus pratensis* L.) also may serve as an agent of inoculation for

celery heart rot. The insects were allowed to feed on celery plants that had been sprayed with a water suspension of *Erwinia carotovora* and incubated in high humidity. Under these conditions, infection occurred in the feeding punctures within 48 hours. Control plants treated in the same way but not subjected to the insects remained healthy except where artificial wounds were present. Since plants sprayed with sterile water and subjected to the insects remained healthy, one would conclude that under natural conditions the tarnished plant bug would be less effective as a vector than the Dipterous insects.

Bacterial Wilt of Cucurbits.—Cucurbit wilt was first described by Erwin F. Smith in 1893. It is confined to the family Cucur-



FIG. 79.—A squash plant affected with bacterial wilt, a disease transmitted in nature only by two species of cucumber beetle.

bitaceae but apparently does not affect all members of the family. Cucumbers are most frequently affected, followed in order of susceptibility by cantaloupes, summer squash, winter squash, and pumpkins. Watermelons are said to be immune or very highly resistant. Bacterial wilt is widespread throughout the eastern part of the United States and has been reported from Europe, South Africa, and Japan. Its prevalence varies from year to year, and it is often very destructive, killing more than half the plants in some fields. Affected plants droop as from the lack of water (Fig. 79). One or more runners may wilt depending upon the point of inoculation. In early stages of infection the plants may droop during mid-day and revive over night only to droop more severely the next day and collapse completely.

Bacterial wilt is caused by *Erwinia tracheiphila* (Erw. Smith) Winslow *et al.*, a white, viscid, capsulate bacillus. The pathogen is a strict vascular parasite, being confined almost entirely to the vascular bundles of affected stems. The white viscid mass of bacteria that exudes from the vascular bundles, when an

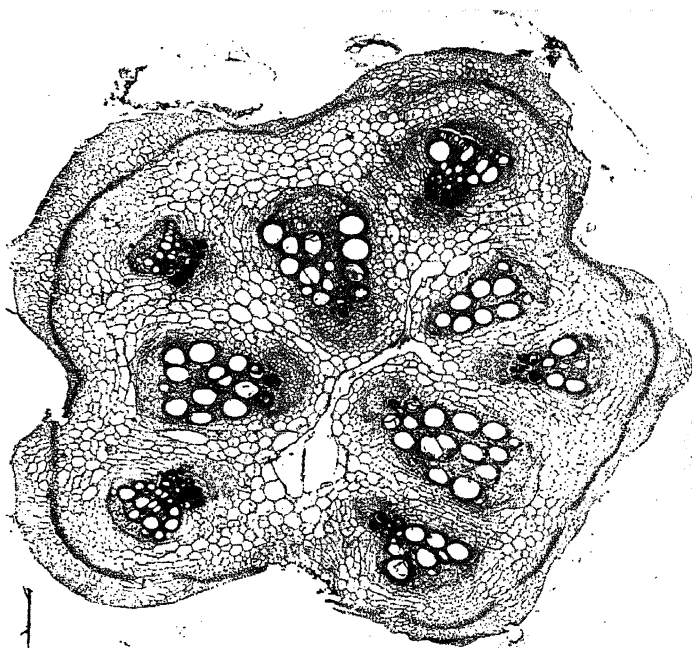


FIG. 80.—A cross section of a cucumber stem infected with bacterial wilt showing the spiral vessels occluded by masses of *Erwinia tracheiphila*. Approx. 12 \times . (Photograph courtesy Bureau of Plant Industry, U. S. Department of Agriculture.)

infected stem is cut, is the best diagnostic symptom of the disease. Wilting is caused largely by occlusion of the tracheal tubes (Fig. 80).

E. F. Smith in 1911 expressed belief that the disease is disseminated by insects, especially the striped cucumber beetle (*Diabrotica vittata* Fabr.) (Fig. 81). The first experimental proof was submitted in 1915 by Rand, who incriminated also the

twelve-spotted cucumber beetle (*D. duodecimpunctata* Oliv.) (Fig. 82). These two insects are the only known vectors of the disease, and no other natural means of spread has been reported. Rand and Enlows (1916) have shown by careful experiment that infection does not come through soil or seed and that the following insects are not vectors: the squash bug (*Anasa tristis* De G.), the squash ladybird (*Epilachna borealis* Fabr.), the melon aphid (*Aphis gossypii* Glov.), the honeybee (*Apis mellifera* L.) and the potato flea beetle (*Epitrix cucumeris* Harris).

Rand and Enlows (1916, 1920) and Rand and Cash (1920) have shown that infection does not occur through the stomata and that wounds involving the vascular bundles are most effective as infection courts. These authors have made extensive studies of the relation of insects to the disease, and most of the available information is from their work. They have shown that the insects serve not only as agents of dissemination and inoculation but that the adult beetle of *D. vittata* (the most common of the two beetles on cucurbits)

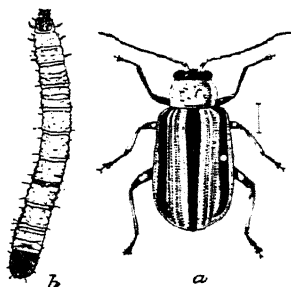


FIG. 81.—The striped cucumber beetle (*Diabrotica vittata* Fabr.), the principal vector of bacterial wilt of cucurbits. a, adult beetle; b, larva. Approx. 5X. (After Chittenden.)

may harbor the pathogen over winter in its body. Primary infection in the spring always originates from the feeding punctures of such overwintered beetles. Not all beetles harbor the pathogen. It was recovered experimentally from a relatively small percentage of the overwintered beetles tested, but only a small number would be necessary to establish centers of infection for secondary spread. Confirmatory evidence of this method of overwintering has been furnished by Doolittle (1921).

The life of the bacterium in the insect body has not been studied in detail by histological methods, and it is not known whether any symbiotic relationship exists. However, Rand and Enlows (1920) have shown that the bacteria pass uninjured through the intestinal tract of the beetles and may be recovered from the feces. Rand and Cash (1920) have demonstrated experimentally that the feces from infective beetles may serve as effective inoculum only if they are dropped into a fresh feeding

wound. Observations indicate that such contaminated feces may be important as inoculum at night when the plants are covered with dew or in rainy weather.

The possible association of the pathogen with the larvae of the beetles has not been investigated adequately. Since the larvae commonly feed on the roots and make wounds in the vascular system, it is entirely possible that they also may be vectors of the disease. When larvae feed on infected plants, they in all probability ingest the pathogen, but the larvae usually do not migrate from plant to plant. The fate of these bacteria in the

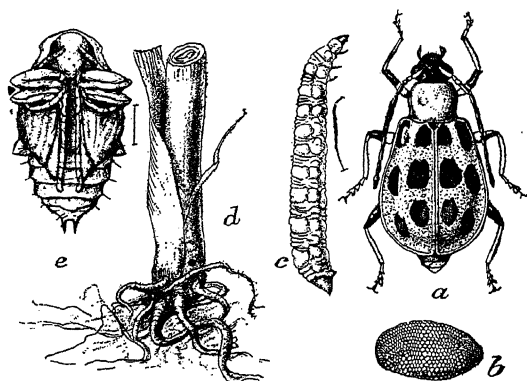


FIG. 82.—The spotted cucumber beetle (*Diabrotica duodecimpunctata*), a vector of bacterial wilt of cucurbits. The larva of this insect is also an important vector of the bacterial wilt of corn. *a*, adult; *b*, egg; *c*, larva; *d*, work of larva at base of a corn stalk; *e*, pupa. (After Chittenden.)

intestinal tract of the larva should be determined. It should be known whether the bacteria survive through the pupal stage and appear in the body of the emerging adult.

Bacterial Wilt of Corn (Stewart's Disease).—The bacterial wilt of corn was described in 1897 by Stewart, who had first observed it on Long Island, N. Y., in 1895. It is primarily a disease of sweet corn, although field corn, teosinte (*Euchlaena mexicana*), and *Tripsacum dactyloides* are known to be susceptible. Wilt is caused by a yellow, nonmotile bacterium [*Phytomonas stewarti* (Erw. Smith) Bergey *et al.*]. Ivanoff (1933, 1935) has reported that other bacteria may cause symptoms similar to those caused by this organism. This species is, however, the most prevalent

and the most important. The pathogen is primarily a vascular parasite although other tissues are often affected. Long, greenish-yellow lesions following the veins are formed on the leaves (Fig. 83). These later turn brown and dry. The entire plant eventually wilts and may die in any stage of development (Fig. 84). The yellow bacterial exudate that comes from the vascular bundles when the stalk is cut in cross section is an important diagnostic symptom (Fig. 85).

Wilt is known only in North America and is most prevalent in the eastern and southern part of the United States. The disease occurred only sporadically in the years following its



FIG. 83.—A corn leaf showing wilt lesions that originated at the feeding wounds made by the corn flea beetle. Note the tendency of the lesions to follow the course of the veins. (After Elliott.)

discovery, but in 1932 and 1933 it became epiphytotic throughout the southeast section of the corn belt. In these years, the disease was very destructive, especially on the early varieties of sweet corn, and numerous reports of 100 per cent losses were recorded. Renewed interest in the disease has led to the discovery of new and important facts, especially concerning the relation of insects to its spread and development.

The early work of Stewart (1897) and Smith (1914) led them to believe that the disease was primarily seed-borne, although infection on aboveground parts was recognized. Natural internal infection as well as surface contamination of seed produced on infected plants was demonstrated by Smith (1914), and it was assumed that the amount of seed infection would determine the amount of disease in a given crop, although the possibility of direct infection from infested soil was not overlooked. Rand and Cash (1921) could find no evidence of infec-

tion from the soil, and similar results were reported in the same year by Reddy (1921). Rand and Cash noted also that infected seed when planted in different regions and at different times gave widely differing amounts of disease, indicating some unknown source of infection.



FIG. 84.—Young corn plants affected with bacterial wilt. The two center plants are heavily infected and partly wilted. The plant on the extreme left is lightly infected, and the one on the right is healthy.

The first proof of insect dissemination of the disease was presented by Rand (1923). By means of cage experiments, he demonstrated that the brassy flea beetle (*Chaetocnema pulicaria* Melsh.) was responsible for secondary spread of wilt in mid-summer (Fig. 86). In 1924, Rand and Cash presented more data and proved that the toothed flea beetle (*C. denticulata* Ill.) also is a vector. In addition, they presented circumstantial evidence as a basis for the hypothesis that "the early seasonal incidence of the disease is due largely to the introduction of the

bacterial parasites by insects working at the roots or base of the stems." They concluded that the probabilities were strongly in favor of primary infection being induced by the wounds made by the southern corn rootworm (*Diabrotica duodecimpunctata* Oliv.) (Fig. 82). A more complete report was published by Rand and Cash in 1933 in which this hypothesis was verified by experimental evidence.

The discovery and recognition of the association of flea beetles with bacterial wilt probably explains the unusually severe type of injury often attributed to these insects of corn. Forbes (1905) reports the wilting of corn plants attacked by flea beetles in

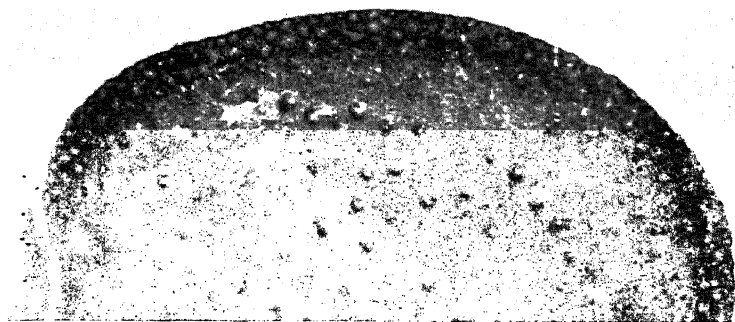


FIG. 85.—A cross section through a corn stem affected with bacterial wilt showing the bacteria oozing from infected vascular bundles. 6X. (Courtesy of Bureau of Plant Industry, U. S. Department of Agriculture.)

Illinois in 1891, and Metcalf and Flint (1928) describe flea-beetle injury on corn as follows: "Growth is retarded, and the leaves wilt even during wet weather." In all probability, bacterial wilt was present on these plants but was not recognized.

Ivanoff (1933) showed that, although the pathogenic bacteria were carried in the chalazal tissues of infected seed, the embryo was not infected and the seedlings would not become infected from this source unless wounded. Infection followed injury of the roots of seedlings by white grubs (*Phyllophaga* spp.) and the larvae of the western corn rootworm (*Diabrotica longicornis* Say) and by artificial injury.

Elliott and Poos (1934) demonstrated that the pathogen commonly survives the winter in the intestinal tracts of the brassy

flea beetle (*C. pulicaria* Melsh.). Overwintered adults caught in April were found to have large quantities of the pathogenic bacteria in their intestinal tracts; and when beetles from the same collection were allowed to feed on healthy corn plants in the greenhouse, typical wilt was produced. Approximately 19 per cent of the beetles studied were found to carry the pathogen, but this species was the only one of a large number of insects tested that harbored the bacteria. The histological details of the survival in the body of the insect have not been reported. The

importance of insects in the epiphytology of wilt has been discussed in considerable detail by Elliott (1935).

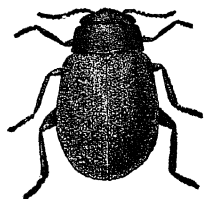


FIG. 86.—The corn flea beetle (*Chaetocnema pulicaria*). Approx. 20 X. This insect is the principal vector of the bacterial wilt of corn. The bacteria causing the wilt live over winter in the bodies of hibernating beetles. (From Illinois State Natural History Survey.)

Frutchey (1936) has demonstrated that the seed-corn maggot [*Hylemyia cilicrura* (Rond.)] (Fig. 71) also may play an important role in the development of the disease. The larvae of this insect were found in a large number of diseased plants in the field. When adult flies were caged on corn plants, eggs were deposited, and the larvae attacked the roots and crown of the plants. Sixty-seven out of 93 plants, or 72 per cent, became infected with wilt, and every wilted plant showed maggot injury. Only an occasional wilted plant was found in the controls. This experiment, however, does not prove whether the insect is a vector or merely an agent of ingress. The mere wounding of the plants in the presence of the pathogen would account for the observed results. In view of the importance of this insect in the development of the bacterial soft rots and its known symbiotic relations with bacteria (Leach 1931*b*, 1933), its role in the development of this disease should be more thoroughly investigated.

Further study of the association of all these insects with the bacteria is needed. The bacteria are known to live for some time within the body of the adult beetles of the southern corn root-worm (*D. duodecimpunctata*), but Rand and Cash (1933) think this insect is of less importance in the spread of midsummer infection than the flea beetles. It is known that the larvae make the wounds necessary for infection through the base of the plants, but their significance in dissemination of the pathogen is not

known. In view of the fact that bacteria are known to survive through pupation in some insects and in some cases are disseminated on or in the eggs, it is of vital importance that these relationships be studied in detail.

Stevens (1933, 1934, 1937) has discussed the relation of winter temperatures to the abundance of corn wilt the following summer, pointing out that the disease appears to be worse following mild winters. If it can be shown that there is a direct and consistent relationship between winter temperatures and the prevalence of the disease in the following summer, it would provide a reliable means of predicting epiphytotics and avoiding losses. Since there appears to be no direct influence of winter temperatures on the disease, its indirect influence on the prevalence of insects which harbor the disease over winter, and on which the bacteria depend for ingression, appears to be a probable explanation. More complete information, however, is necessary before this can be accepted as the true explanation. Support for this theory is found in the observations reported by Elliott (1938) in which the prevalence of the disease in the northeastern states in 1938 was closely correlated with the prevalence of the flea beetle (*C. pulicaria*). In this year, the prevalence of the disease increased rapidly in early summer but decreased strikingly in late summer. The beetles also were abundant until the middle of July but decreased rapidly until very few could be found in late August.

Haenseler (1937) made a similar study of the disease based on records from 1910 to 1937 in New Jersey. He confirmed Steven's conclusions in general but expressed the belief that the relationship was not so simple that reliable predictions could be made without further study. He showed there was evidence of a lag period following a severe winter before the full effect is felt. A similar lag period following a mild winter indicated that more than one year was necessary for building up the disease to epiphytotic prevalence. It should also be recalled that insect parasites and other factors may influence the prevalence of the vectors of wilt.

Wellhausen (1937) has demonstrated a modification of the virulence of the wilt pathogen as a result of passage through susceptibles of differing degrees of resistance, the strain becoming less virulent after passage through very susceptible hosts. It

was suggested that this phenomenon might influence the epiphytology of the disease. The introduction of wilt-resistant varieties of sweet corn is contributing much to the practical control of the disease (G. M. Smith 1933, Ivanoff and Riker 1936), and the variety Golden Cross Bantam and other resistant hybrids are rapidly replacing the more susceptible varieties (Stevens 1937).

Olive Knot.—The olive is subject to a bacterial disease causing “knots,” or galls, on the branches of the tree (Fig. 87). The



FIG. 87.—Olive knot caused by *Phytoplasma savastanoi*. A, a heavily infected tree; B, a single small branch with seven knots. (After Wilson.)

disease is prevalent in Italy and other countries of southern Europe and has been known in California since 1898 (Wilson 1935). Olive knot is caused by *Phytoplasma savastanoi* (Erw. Smith) Bergey *et al.* Infection is known to occur commonly through wounds made by freezing, pruning operations, or other agencies and also through leaf scars (Horne, Parker, and Daines 1912; Wilson 1935). Rain water appears to be the principal agent of inoculation in California and probably elsewhere, but Petri (1909, 1910) has reported that in Italy the disease is closely associated with the olive fly (*Dacus oleae* Rossi). He

has described a highly developed symbiotic relationship between *D. oleae* and certain species of bacteria. These bacteria are constantly found in the intestinal tract of the insect in all stages of development. The bacteria are transmitted internally through the egg and survive in the puparium. Striking anatomical modifications are found in the insect which ensure perpetuation of the bacteria through successive generations. Among the bacteria frequently occurring as a symbiote is a nonpathogenic species (*Ascobacterium luteum*), but according to Petri

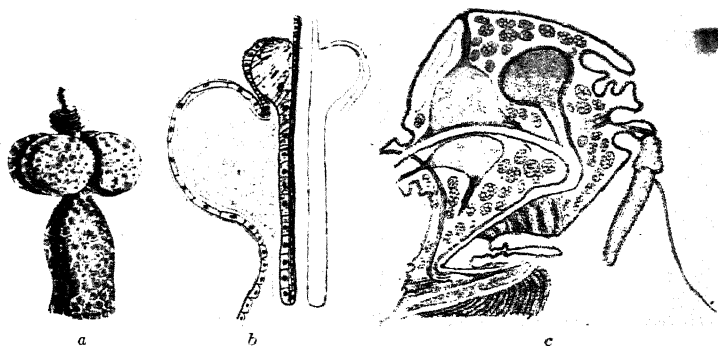


FIG. 88.—*a*, the anterior portion of the mid-intestine of the larva of *Dacus oleae*, in perspective, showing the caeca filled with bacteria; *b*, the same as seen in section; *c*, a longitudinal section of the head of an imago of *Dacus oleae* showing the bulbous diverticulum off the esophagus. During metamorphosis, the bacteria from the intestinal tract accumulate in this organ from which the entire intestinal tract is later recontaminated. (After Petri.)

the olive-knot pathogen also is usually present. Although it is difficult to isolate in pure culture directly from the insect, its presence can be demonstrated by inoculating olive branches with the mixed cultures. The histological aspects of this symbiotic association have been verified by Stammer (1929) who also has described in other species of Trypetidae a series of symbiotic relationships with anatomical adaptations of various degrees of complexity.

Because of the highly developed symbiotic relationship pictured here, the association will be described in some detail. The principal features have been graphically illustrated by Petri as shown in Figs. 88, 89, and 90. Petri has demonstrated that

the intestinal tract of the fly is constantly contaminated with the bacteria, some of which pass out uninjured in the feces. In the female fly, the anal tract and the vagina unite at their posterior end to form a common opening to the exterior (Figs. 89 and 90). In the wall of the anal tract near the point of union, there are a number of saclike evaginations that open into the lumen. These

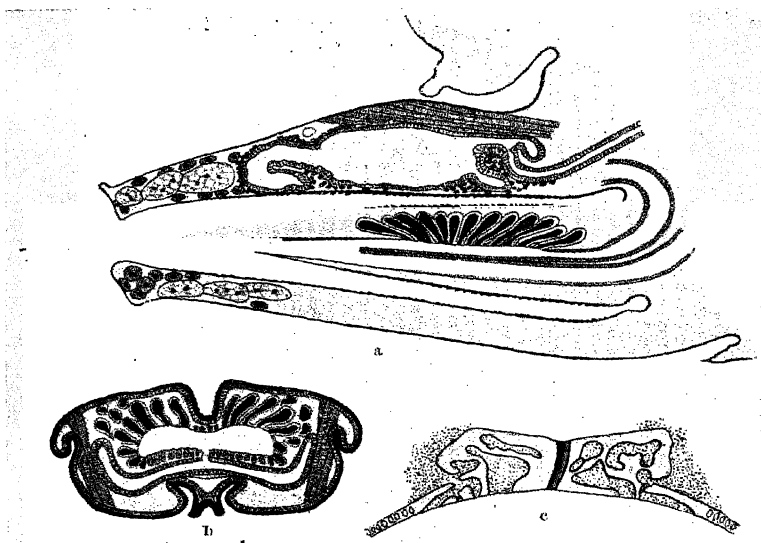


FIG. 89.—The ovipositor of *Dacus oleae*, a vector of olive knot showing the anatomical adaptation for congenital transmission of bacteria. *a*, a longitudinal section of the ovipositor showing the common opening of the vagina and the anal tract and the saclike evaginations of the anal tract in which the bacteria are found; *b*, a cross section of the ovipositor showing the longitudinal slit connecting the anal tract and the oviduct (the eggs on passing out press against the pockets and become smeared with bacteria); *c*, a section through the micropyle of an egg through which the bacteria enter the egg to infect the embryo. (After Petri.)

are always filled with bacteria. Immediately opposite the opening of the evaginations there is a longitudinal slit in the membrane, separating the anal tract from the oviduct. When the eggs pass along the vagina, this slit is spread open so that the surface of the egg is pressed against the openings of the bacteria-filled evaginations. In this way the bacteria are smeared over the surface of the egg.

Petri has shown that the bacteria find their way through the micropylar openings into the interior of the eggs and into the body of the developing embryos (Fig. 89c). The larvae are, therefore, always internally contaminated with the bacteria before they hatch from the egg. Near the fore part of the mid-intestine of the larva are four spherical caeca that harbor the bacteria (Figs. 88a and b) which may be found distributed through-

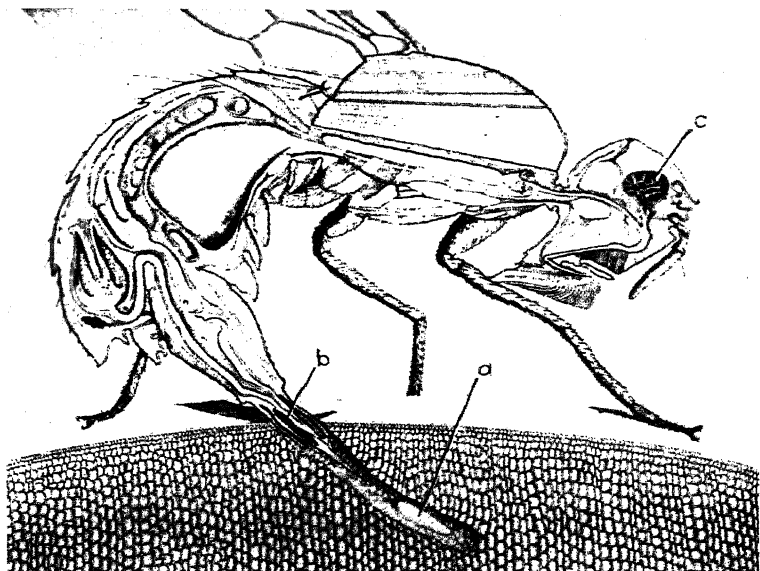


FIG. 90.—A diagrammatic sketch showing how the eggs of *Dacus oleae* are deposited and how the tissues of the plant are inoculated with bacteria. a, egg; b, saclike evaginations of the anal tract filled with bacteria; c, diverticulum of esophagus filled with bacteria. (After Petri.)

out the contents of the intestinal tract. When the larva pupates, the bacteria decrease in number but do not entirely disappear; for when the fly emerges from the puparium, it is always internally contaminated. A peculiar spherical diverticulum is developed off the esophagus just in front of the brain of the pupa (Fig. 88c). This diverticulum soon becomes filled with the bacteria. Shortly after the imago emerges from the puparium, the entire intestinal tract, including the anal sacs, becomes contaminated from this reservoir. By this specialized adapta-

tion, the perpetuation of the symbiotic bacteria is ensured. The bacteria are inserted into the wounds made in oviposition (Fig. 90), and according to Petri such wounds are infection courts for much of the olive knot in Italy. In California, where this insect does not occur, the bacteria depend chiefly on wind-blown rain for spreading from tree to tree, and the rate of spread from a center of infection is relatively slow (Wilson 1935). It is entirely probable that the spread would be more rapid if the insect were present.

Stammer (1929) has shown by histological studies that many species of Trypetidae harbor symbiotic bacteria. It has been shown (Allen and Riker 1932) that the Trypetid apple maggot

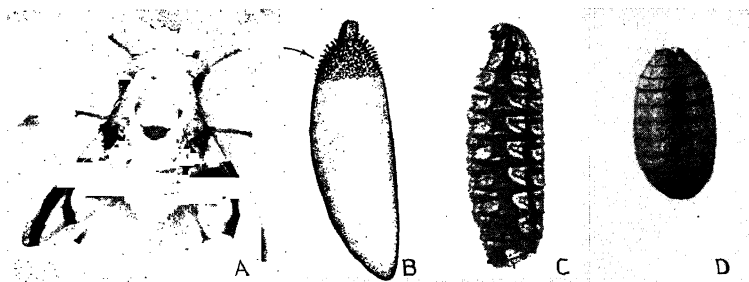


FIG. 91.—The apple maggot fly (*Rhagoletis pomonella*). A, adult female; B, egg; C, larva; D, puparium. (After Allen, Pinckard, and Riker.)

(*Rhagoletis pomonella* Walsh) is a regular vector of a bacterial rot of apples and that there is a close symbiotic association between the insect and the pathogenic bacteria. Whenever a representative of this group of insects is found associated with a bacterial disease, it should be considered as a potential vector.

Bacterial Rot of Apples and the Apple Maggot.—Although many people have observed that a decay of apples often followed infestation by the apple maggot (*Rhagoletis pomonella* Walsh) (Fig. 91), no particular significance was attached to the association. It was generally assumed that the breakdown of the tissue (Fig. 92) was due to the activity of the maggots alone or to chance entrance of pathogens through the wounds made by the insects. Allen (1931) reported that the decay associated with maggot injury was caused by bacteria and suggested that they were disseminated by the adult flies. Allen and Riker (1932)

published a more complete account of the decay, described the pathogen, and named it *Phytomonas melophthora*. In 1934, Allen, Pinckard, and Riker described in considerable detail the association of the bacteria with the various stages of the life cycle of the apple maggot.

The female fly deposits her eggs in the apple fruit by making a puncture through the epidermis with her sharp pointed ovipositor (Fig. 93). The eggs hatch in 2 to 9 days; the larvae tunnel through the flesh of the apple and, aided by the bacteria



FIG. 92.—Apples showing decay associated with larval tunnels of the apple maggot. The decay is caused by *Phytomonas melophthora* and is transmitted exclusively by the apple maggot. A, surface view showing decay originating at oviposition punctures (h); B, section showing internal decay and larval activity. (After Allen and Riker; and Allen, Pinckard, and Riker.)

with which they inoculate the apple, soon render the fruit worthless. The length of the larval period varies from 2 or 3 weeks to several months. The rate of development is slower in green apples than in ripe ones. When the maggots are mature, they leave the apples and enter the ground to pupate. The insect lives over winter in the pupal stage and emerges the following summer.

The bacteria are found associated, externally and internally, with both male and female flies. They also were isolated in about 50 per cent of the trials from oviposition punctures and equally as often from the surface of eggs removed from apple

tissue. Two larvae that hatched from surface-sterilized eggs were sterile, a fact indicating that the bacteria do not occur inside the egg but that they have ample opportunity of becoming contaminated by the bacteria borne on the surface of the eggs.

The tissue of green apples appears to be resistant to the bacteria, which cause very little decay before the fruit begins to ripen.

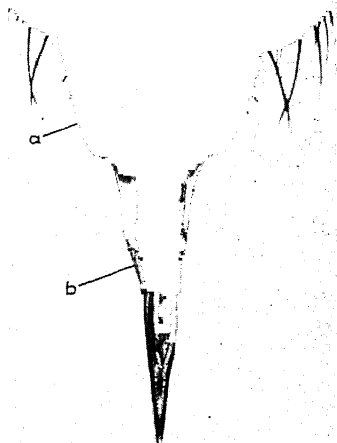


FIG. 93.—The ovipositor of the apple maggot fly partly extended. *a*, end of abdomen; *b*, tip of ovipositor. By means of this sharp pointed structure, the fly punctures the apple tissue and inserts an egg, at the same time inoculating the apple tissue with pathogenic bacteria carried on the surface of the egg. 23 X. (After Allen, Pinckard, and Riker.)

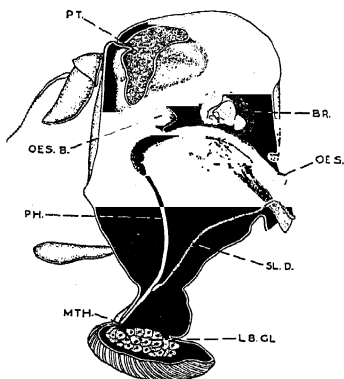


FIG. 94.—A section through the head of an apple maggot showing, among other structures the "oesophageal bulb" (OES B). Its function is not known, although a similar structure in the olive fly (*Dacus oleae*) harbors symbiotic bacteria during metamorphosis. *PT*, ptilinum; *BR*, brain; *OES*, oesophagus; *PH*, pharynx; *SL. D*, salivary duct; *MTH*, mouth; *LB. GL*, labial gland. (After Dean.)

The development of the larvae may be dependent upon the bacterial growth, for the maggots appear to prefer the decayed tissue. Two sterile maggots failed to live in apple tissue, while maggots from nonsterilized eggs grew and pupated normally. No further studies on the nutritional significance of the bacteria have been reported.

Bacteria were not isolated from the interior of the puparia although they were abundant on its surface. It was mentioned that considerable difficulty in isolation of the pathogen was

caused by contaminating bacteria. In view of the frequency of the survival of bacteria in the puparia of other Diptera (Stammer 1929, Leach 1933, Ark and Thomas 1936), it would appear that this aspect of the association needs further investigation.

No histological studies were reported by Allen, Pinckard, and Riker; but Dean (1933, 1935) has reported on certain phases of the morphology and anatomy of the apple maggot. There are

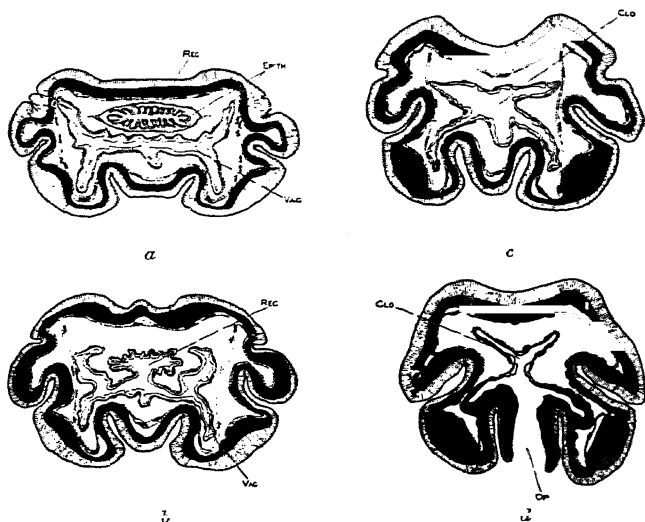


FIG. 95.—Cross section of the ovipositor of the apple maggot fly at four points near the posterior end, showing the union of rectum and vagina. *a*, the most anterior section showing the rectum (*REC*) and vagina (*VAG*) separate. Note the numerous diverticula in the epithelium (*EPITH*) of the rectum; *b*, a more posterior section showing the rectum and vagina beginning to unite; *c*, the rectum and vagina completely united into a common cloacal passage (*CLO*); *d*, the external opening of the cloaca (*OP*). Approx. 200 X. (After Dean.)

certain points of similarity in the anatomy of this insect with that of *Dacus oleae* which are significant. A peculiar evagination of the esophagus similar in all respects to that described in *D. oleae* by Petri (1909, 1910) was reported by Dean (Fig. 94), who apparently was not familiar with the work of Petri and made no attempt to determine the function of this structure which he called the "esophageal bulb." It should be determined whether or not the bacteria that survive through metamorphosis occupy this organ as they do in *D. oleae*.

There also is a striking similarity in structure of the ovipositor and anal tract of the two insects. Dean reported that the rectum and vagina of the apple-maggot fly unite to form a common passage. His illustrations show the same type of evaginations of the anal tract to form numerous sacs similar to those described with respect to *D. oleae* by Petri although no special mention is made of them (Fig. 95). A closer study of these structures with particular reference to their association with symbiotic bacteria should be made. In all probability, it will be found that the pathogenic bacteria are transmitted on or in the egg as in *D. oleae*.

Gummosis of Sugar Cane.—One of the more destructive diseases of sugar cane is that known as “gumming” or “gummosis.” It is caused by a bacterium [*Phytomonas vascularum* (Cobb) Bergey *et al.*] that develops primarily as a vascular parasite causing copious exudates of a sticky gum, a characteristic that is responsible for the name of the disease. The disease has been known since 1869 but was first adequately described by Cobb (1893, 1895), who showed that it was caused by a bacterium, which he named *Bacillus vascularum*. The pathogen was more completely described and its pathogenicity verified by R. G. Smith (1902) and by E. F. Smith (1904). The most thorough study of the disease and its method of spread under field conditions has been made by North (1935).

Gummosis of sugar cane is strictly confined to the sugar-cane plant. No other plant is known to be affected in nature, although there is some evidence that the disease may have originated in South America where sugar cane is not indigenous. If this is true, it would be necessary to assume its pathogenicity on some plant native to South America.

Affected plants are dwarfed, and long yellow streaks appear on the leaves. The streaks later wither and become brown, the leaves dying from the tip toward the base. A pronounced yellow slime that forms in the vascular bundles is the best diagnostic symptom. The infection spreads from the vascular bundles into the neighboring cortex, and in wet weather a yellowish gummy exudate forms on the surface of affected tissue.

Gummosis is transmitted readily by planting infected sets; and, as sugar cane is commonly propagated in this way, this method of transmission is very important. It is the chief method of introducing the disease into new areas and has been responsible

for most of the long-distance spread. The disease also may be transmitted by the cutting knife when canes are being cut for planting.

Local secondary spread in the field is effected primarily by wind and rain as demonstrated by North. Gum, filled with the pathogenic bacteria, oozes from affected leaves when they are wet. Rain washes the bacteria from the leaves and scatters it to other leaves which are at the same time being injured by the effects of the wind. Infection occurs most commonly through minute wounds made by the serrate edges of one leaf scratching the surface of another leaf.

North (1935) made a thorough study of insect transmission of gummosis under field conditions. In an extensive series of cage experiments, only negative evidence was obtained with numerous species of insect that commonly feed on sugar-cane leaves, including aphids, leaf hoppers, plant bugs, and beetles. However, many species of fly were observed actively feeding on the gummy exudate, especially in wet weather following a rain. It was demonstrated by a series of caging experiments that flies were effective vectors. The flies take shelter in the sugar-cane fields during the rainstorms, and when the storms are over they are attracted to the sap oozing from the fresh wounds. They ingest contaminated gum from infected plants and then feed on exudate from fresh wounds in healthy plants and in this way transmit the disease. It was also demonstrated that the bacteria survive for some time in the intestinal tract of the flies and that the flies may transmit the pathogen for relatively long distances as compared to the local dissemination by wind-blown rain. The importance of transmission by flies is based primarily on relatively long-distance dissemination that gives rise to isolated centers of infection and secondarily on local dissemination as a supplement to dissemination by wind and rain.

Control of gummosis depends largely upon the selection of healthy canes for planting, the use of general sanitary practices, and the development and planting of resistant varieties. Some progress has been made in the development of resistant varieties; and this method offers the most promise, for the other methods are effective only to a limited extent.

A Bacterial Disease of Willows and the Willow Borer.—Lindeijer (1932) has described a disease of willows in Holland

caused by a species of bacteria which she listed as a new species (*Pseudomonas saliciperda*), [*Phytomonas saliciperda* (Lindeijer) Hauduroy *et al.*]. The willow borer, or snout beetle (*Cryptorhynchus lapathi* L.), is reported as a vector of the disease.

The disease causes a wilt of the branches followed by early defoliation and death of the affected limbs, either the first or second year after infection (Fig. 96a). The pathogen is a vascular parasite. It is a capsulate organism that clogs the vessels and cuts off the transpiration stream. The terminal portions of

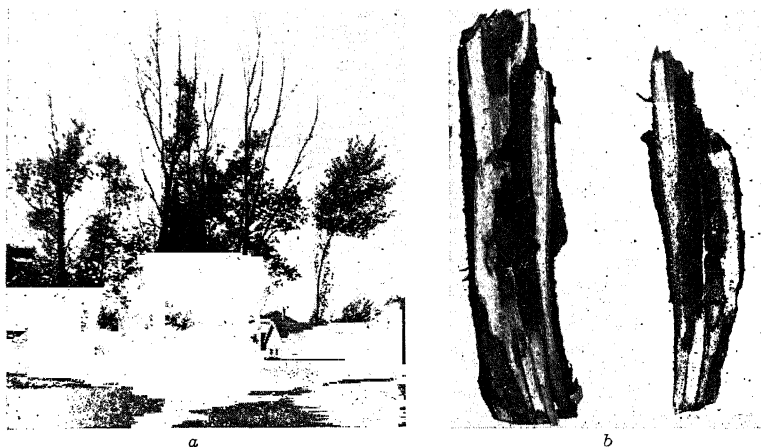


FIG. 96.—Bacteriosis of willow. *a*, a group of willow trees killed by bacteriosis; *b*, two twigs with burrows made by *Cryptorhynchus lapathi*, the vector of the disease. (After Lindeijer.)

the branches dry out, whereas the lower parts become waterlogged. Infection can occur only through fresh wounds that penetrate the bark and reach vessels of the wood.

The necessary wounds are usually furnished by the feeding and breeding activities of the willow borer. This insect feeds upon the bark and the outer layers of the wood. Observations showed that natural infections most frequently originated at the wounds made by the insect. The borer feeds readily upon infected branches and in so doing becomes contaminated with the bacteria (Fig. 96b). The disease was experimentally produced by allowing insects, both naturally and artificially con-

taminated with the bacteria, to feed upon willow twigs. The bacteria also were isolated from the esophagus of beetles that had fed upon infected branches.

The borer also breeds in willow twigs, but eggs deposited in infested wood rarely hatch because of the excessive moisture. It is concluded, therefore, that the vector does not as a rule become contaminated from breeding in infested wood although it may be contaminated from walking over the bacterial ooze that is often exuded from cracks in affected branches.

Bacterial Wilt of Solanaceae.—Potatoes, tobacco, tomatoes, peppers, and other related plants are severely affected with a bacterial wilt caused by *Phytomonas solanacearum* (Erw. Smith) Bergéy *et al.* Numerous plants in families other than the Solanaceae also are affected to some extent. It is prevalent throughout the southern part of the United States and has been reported from Europe and several places in the tropical regions of the world. It appears to be favored by warm weather and is most destructive in warmer regions.

The first symptom of the disease is a sudden wilting of the leaves on a single branch or on the entire plant. This is soon followed by a shriveling of the stems and eventually the death of the plant. The bacteria are found first in the vascular bundles which become brown throughout almost the entire plant, but eventually they enter the parenchyma cells of the cortex and pith and destroy them as well. The bacteria spread through the vascular bundles of the stolons until they reach the tubers where the vascular ring is first destroyed, and the decay of the storage tissues soon follows. When affected stem or tubers are cut in cross section, the bacteria ooze out in a brownish yellow slime.

From a study of the literature on this disease, it is evident that many details of the methods of infection and spread in the field are not known definitely. Smith (1896) succeeded in transmitting the disease in the greenhouse by means of potato beetles (*Leptinotarsa decimlineata* Say). Potato beetles, having previously fed on infected plants, were placed for several hours on four healthy plants under large bell jars after which they were removed. Within 7 to 9 days, all the inoculated plants developed typical symptoms, while the controls remained free. From these experiments, Smith generalized as follows:

These experiments with the Colorado potato beetle seem to fully warrant the conclusion that insect enemies are largely responsible for the spread of the disease. . . . Given one diseased vine in a field and plenty of insects to feed upon it, the transmission of this disease to all parts of the field, and thence to the whole neighborhood, is only a question of weeks.

Just what insects are most instrumental in disseminating this parasite in any particular locality can be determined only after a prolonged and careful study of the disease in the field. No experiments have been made with other insects, but it is likely that flea beetles, blister beetles, chrysomelids, and many other leaf-eating insects may act as carriers of the disease.

These general conclusions have been repeated by subsequent writers, but the "prolonged and careful study of the disease in the field" seems never to have been made.

Although Smith (1896) concluded that most of the infections probably occur aboveground as the result of insect injuries, his views were modified later (1914), and much circumstantial evidence has been presented by various workers, indicating that in nature infections occur most commonly on the underground parts. The recent work of Eddins (1936), in which effective control of the disease on potatoes was obtained by adding sulphur to the soil, lends weight to the latter conclusion.

The available evidence indicates that infection occurs only through wounds. At least, it has never been demonstrated that infection can occur in the absence of wounds. If wounds on the underground parts are necessary for infection it is very probable that insects are involved. A thorough study of the soil insects that commonly affect the roots of susceptible plants would probably throw some light on the question. Smith (1914) has suggested a possible relationship with the root-knot nematode, but no experimental study of the relationship has been reported.

Bacteriosis of Prickly-pear Plants.—Johnson and Hitchcock (1923) have described a bacterial disease of prickly pear (*Opuntia spp.*) which is transmitted by a number of insects that feed upon these plants. The disease was discovered in Florida and later introduced into Australia where it was studied as a possible aid in the control of prickly pears.

The disease is caused by a species of bacteria described and named by Johnson and Hitchcock as *Bacillus cacticidus*, [*Erwinia*

cacticida (Johnson and Hitchcock) Hauduroy *et al.*]. The bacteria cause a rapidly developing soft rot of affected tissues. The decay is limited almost entirely to the succulent parenchyma tissues, the bacteria failing to spread through the vascular bundles. A distinct purplish discoloration is characteristic of the affected tissues in the earlier stages of decay. The parenchyma is ultimately reduced to a brown, slimy, fetid liquid that dries out leaving only the cuticle and vascular strands.

Inoculation experiments with the pathogen revealed a high degree of specificity for the various species of *Opuntia*. The most commonly grown fruits, vegetables, and forage crops are not susceptible to the disease. The pathogen is an actively motile, Gram-negative, aerobic and facultative anaerobic short rod. In general character, it resembles very closely the omnivorous *Erwinia carotovora* but is more limited in its host range.

The pathogen will not infect through the stomata and is, in all probability, a strict wound parasite. It was demonstrated experimentally that the larvae (caterpillars) of several insect pests of the cactus, including *Melitara prodenialis*, *Olyca junctoliniella*, and *Mimorista flavidissimalis*, are effective vectors of the disease, and it was predicted that *Cactoblastis cactorum* and *C. bucyrus* would also prove to be effective vectors. The larvae of the longicorn beetles (*Moneilema spp.*) and several members of the Drosophilidae also were shown to be capable of transmitting the disease.

The insect vectors of this disease are important not only in transmitting the disease from plant to plant but also in transmitting infection from one segment to another of the same plant. The infection will not spread unaided from one segment to another, for the bacteria affect only the parenchyma tissues and are unable to invade the lignified vascular tissues. Without the aid of the insect vectors, the disease, at most, would produce only local lesions on isolated leaf segments.

It is not known to what extent this bacterial disease has contributed to the destruction of prickly pears in Australia (see Figs. 5 and 6), but Dodd (1936), in discussing the manner of destruction of prickly pears by the introduced insect (*C. cactorum*) stated:

The larvae of *Cactoblastis* devour the whole of the cladodes, except the vascular bundles and the thin papery cuticle, and the injured segments

dry out. More often, however, the tunneling of the larvae sets up conditions favorable for plant diseases, bacterial in the more terminal growth, fungus infections in the thicker lower portions; in consequence, destruction is hastened.

Bacterial Gall of Douglas Fir and *Chermes Cooleyi*.—Hansen and Smith (1937) have described, on Douglas fir in California, a bacterial disease that they believe is insect-transmitted. The disease causes rough globular galls on the young twigs and branches (Fig. 97). The gall is composed of hypertrophied

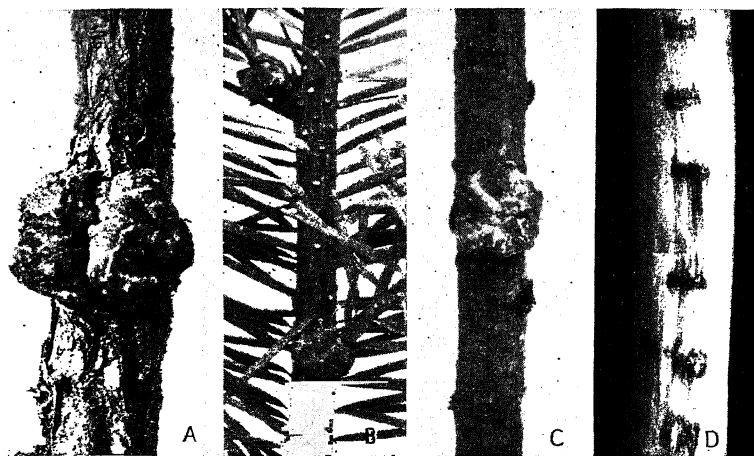


FIG. 97.—Bacterial gall of Douglas fir. A, a naturally occurring gall; B and C, galls produced by artificial inoculation; D, a fir twig with bark removed to show feeding wounds made by *Chermes cooleyi* through which infections occur in nature. (After Hansen and Smith.)

tissue involving both cortex and stele. The galls have their origin always within the tissues of the stele, and artificial inoculations demonstrated that infection occurs only when the pathogenic bacteria (*Bacterium pseudotsugae* Hansen and Smith) are placed in actual contact with the xylem elements.

The dependence of infection upon deep wounds suggested transmission by an insect vector of some sort. Two insects were present in sufficient abundance to invite suspicion. One of these, the orchard cicada (*Platypedia areolata* Uhler), oviposits in the twigs and thus causes deep wounds that reach the xylem elements. Over two hundred oviposition wounds were examined, but galls

were never associated with them. The other possible vector, Cooley's Chermes (*Chermes cooleyi* Gill), is a sucking insect belonging to the family Chermidae. It feeds on the small twigs and makes deep wounds extending into the wood. This insect was observed feeding upon fresh galls, and galls have been found arising from feeding punctures of the insect. Thus strong circumstantial evidence points to this insect as the vector of the disease. No other means of transmission in nature is known.

Black Rot of Crucifers.—As early as 1897, E. F. Smith reported experimental transmission of black rot of crucifers [*Phytophthora campestris* (Pammel) Bergey *et al.*] by slugs (*Agriolimax agrestis* L.) and the cabbage worm (*Plusia brassicae*). Brenner in 1904 confirmed Smith's experiments with slugs and added an unnamed species of aphid as a vector. These experiments were all based on greenhouse work, and no one has shown that the vectors are of any significance in the development of the disease in nature.

In 1924, Clayton reported experiments in which caged cauliflower plants escaped infection while control plants not protected by cages were heavily infected. This led him to conclude that insects were important vectors in the field. Later experiments (1929), however, failed to support this conclusion. The evidence obtained later led to the belief that the cages prevented spread by wind-blown rain and by contact in cultivation, ways in which he concluded the disease was most frequently spread. Other workers have suggested various insects as possible vectors but have presented little or no experimental data. Until more conclusive evidence is available, it must be assumed that in nature insects are of little significance as vectors of black rot.

Angular Leaf Spot of Tobacco and the Southern Tobacco Worm.—Johnson (1934) described an association of the lesions of angular leaf spot [*Phytophthora angulata* (Fromme and Murray) Bergey *et al.*] with injuries made in the leaf surface by the tobacco worm in crawling over the leaf. The prolegs of the tobacco worm are equipped with semicircular hooks and are used for clinging to and moving over the surface of the leaf. These hooks make very small punctures in the leaf that serve as infection courts. The prolegs of the worms doubtless are contaminated with the bacteria from leaf-spot lesions. The insect is more effective as an agent of inoculation when the leaves are moist and is responsible for dissemination for very short distances only.

Bean Bacteriosis and Thrips.—Buchanan (1932) has shown that *Heliothrips femoralis* Reut. may transmit a bacteriosis (halo blight) of beans (*Phytomonas medicaginis* var. *phaseolicola* Burkholder) in the greenhouse. The thrips were observed feeding on plants that had not been inoculated with the bacteria. The bacterial lesions were always associated with the feeding wounds of the insects. When thrips were transferred from infected plants to healthy plants, similar lesions were developed about their feeding wounds, while no lesions developed when the thrips were taken from healthy plants. The transmission appears to be incidental and entirely mechanical. The insect probably is of little importance as a vector of the disease under field conditions.

Blade Blight of Oats (Halo Blight).—Manns, in 1909, reported experimental evidence showing that aphids were vectors of a bacterial disease of oats which he called "blade blight." It is probable that this disease is the same as halo blight caused by *Phytomonas coronofaciens* (Elliot) Bergey *et al.* (Elliot 1920). Aphids, taken from affected plants and transferred to healthy ones in cages, effectively induced the disease after 10 to 12 days, but when the insects were taken from healthy plants no disease developed. Since Elliot has shown that infection may result from stomatal penetration and that aphids are not necessary for infection, it is probable that these insects play only a minor part in the spread and development of the disease. However, it would be well to study the disease further with particular reference to the role of insects in its transmission.

Gardenia Bud Drop.—Wilson (1927) has described a bud drop of gardenias said to be caused by an unnamed species of *Erwinia*. Infection occurs through the extrafloral nectaries. Observations in greenhouses and experiments in the laboratory demonstrated that the bacteria were disseminated by ants and mealy bugs which commonly feed on the extrafloral nectar.

The Spot Disease of Cauliflower and the Red-bordered Stinkbug.—Goldsworthy (1926) in a study of the spot disease of cauliflower [*Phytomonas maculicola* (McCulloch) Bergey *et al.*] isolated the pathogen from the soil and also from the extremities of the red-bordered stinkbug (*Europhthalmus convivus*). He concluded that the insect was a common agent of dissemination although no experimental proof was given.

Bacteria Associated with Aphids and a Gall on Witch Hazel.—Lutz and Brown (1928) have described a species of bacterium commonly associated with the aphid (*Hamamelistes spinosus*) and a spiny, hollow bud gall on witch hazel. They suspect that the bacteria may be the actual cause of the gall but were unable to prove it by artificial inoculation of witch-hazel buds with the bacteria.

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CHAPTER VII

INSECTS AND FUNGUS DISEASES

Fungi are responsible for more plant diseases than any other group of microorganisms. The great diversity in their morphology, physiology, and life history makes them a very versatile group of plant pathogens. The spores, which constitute the principal inoculum of the pathogenic fungi, are extremely varied and are adapted to many different methods of dissemination. Although wind is the most common agent of dissemination of fungus spores, some pathogenic fungi are almost entirely dependent upon insects, not only for spore dissemination but also for inoculation and ingression.

Unlike the bacteria, some species of fungi are able to gain entrance to plant tissues by direct penetration of the epidermal cells. Others depend upon stomata and other natural openings for ingression, but there are some that are completely dependent upon wounds, many of which are made by insects. The adaptive relationships between insects and fungus plants pathogens are as numerous and complicated as those between insects and bacteria.

Ergot of Cereals and Grasses.—Ergot is a fungus disease attacking many species of cereals and grasses. Of the cultivated cereals, rye is the most severely affected. The disease affects primarily the floral organs which often may be so severely injured that the floret is sterile. More frequently, the kernels are replaced by the large, dark-colored sclerotia of the pathogen. The disease is important not only because of the reduction in crop yield but also because of the harmful effect of the sclerotia when eaten by man or animal. The toxic substances in the ergot fungus have certain medicinal properties and are extracted and used as drugs.

The fungus [*Claviceps purpurea* (Fr.) Tul.] survives the winter in the soil as sclerotia. In late spring, the sclerotia germinate by sending up several stromata, each consisting of a light-colored stalk surmounted by a reddish spherical head (Fig. 98).

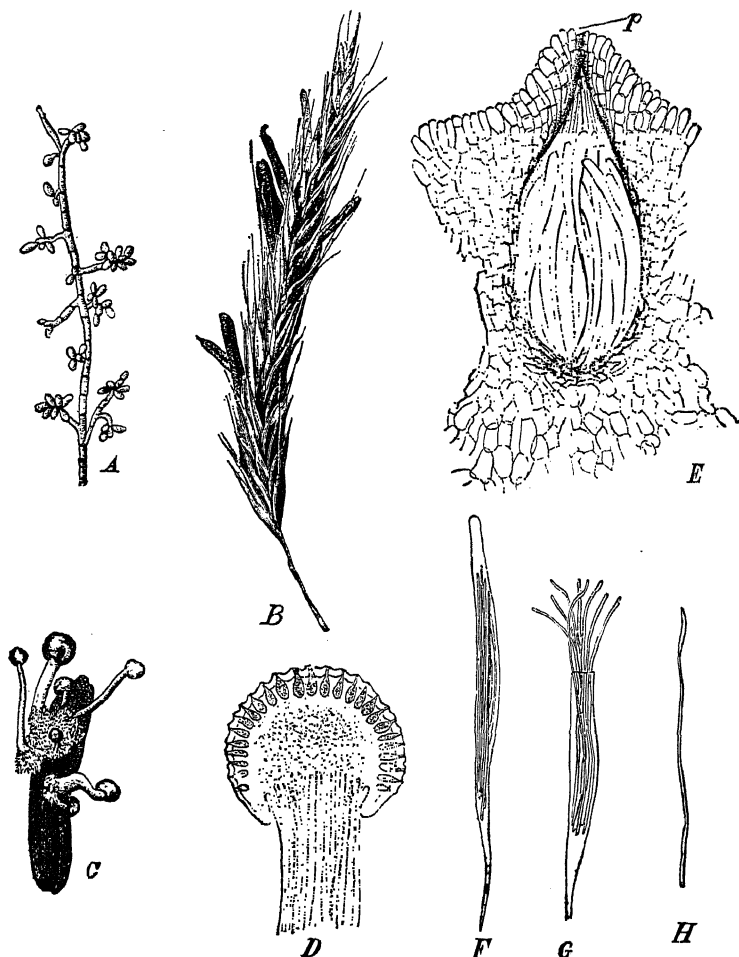


FIG. 98.—Ergot of rye (*Claviceps purpurea*). A, conidia (these are born in drops of a sugary solution on the surface of the young sclerotia); B, mature sclerotia on head of rye; C, a germinating sclerotium with a number of perithecial stromata; D, an enlarged section through a perithecial stroma showing the perithecia embedded near the surface; E, section through a single perithecium; F, a single ascus; G, a ruptured ascus with extruding ascospore; H, a single ascospore. (A and B after Strassburger; C-H after Tulasne.)

Embedded under the surface of the heads are large numbers of flask-shaped cavities opening to the outside through small holes marked by minute papillae. These cavities are the perithecia in which the ascospores are borne. The ascospores are responsible for primary infection.

There has been some difference of opinion concerning the manner in which the ascospores are disseminated to the grass flowers where infection occurs. Stäger (1903) concluded from observations and experiments that the ascospores slowly exude from the perithecia, accumulate on the slimy surface of the stromatic head, and are disseminated by flies. Other workers (Wilson 1875, Falck 1911) have shown that the ascospores are often forcibly ejected from the asci, being projected into the air for a distance of 2 to 8 centimeters, after which they are disseminated further by convection currents and wind.

Because wind dissemination of ascospores has been demonstrated, it is often stated that insects are of no importance in distributing the ascospores of ergot. Such a definite statement does not seem to be justified, for there have been no careful experiments dealing with the subject. As early as 1875, Wilson pointed out that, although some of the ascospores are forcibly ejected into the air, others later are gradually exuded from the perithecia where they collect on the surface of the stroma in a sticky liquid. If insects are attracted to the stroma as reported by Stäger (1910) and if these insects are the same as those which visit the blossoms of rye and other grasses, they would be very effective agents of inoculation. Furthermore, it has been shown (Engelke 1901) that the ejection of the ascospores may occur very suddenly in response to a slight touch of the stromatic head. An insect alighting upon the stromatic head would provide the necessary stimulus and as a result would become contaminated with the ejected spores.

According to Rolfs, as reported by Stevens and Hall (1910), a related fungus (*Claviceps paspali*) is disseminated by beetles of the family *Carabidae*. The beetles visit the spore-covered stromata where they become contaminated with spores, then crawl up the grass stems to the inflorescence, and inoculate the young ovaries. It is evident from the work of Falck (1911) and others that wind dissemination of ascospores occurs quite commonly, and this may be the usual method, but the possibility

of extensive insect dissemination is not excluded. A more thorough study of ascospore dissemination is needed.

Following infection, the ergot fungus permeates the tissues of the ovary, later replacing it with sclerotial tissue. In the early stages of infection, the surface of the developing sclerotium is covered with numerous convolutions on which are borne large quantities of small subspherical conidia (Fig. 98). These spores, measuring from 0.7 to 3.5 microns in diameter, are borne in



Fig. 99.—A, a head of rye showing sugary exudate containing conidia of the ergot fungus; B, flies feeding upon ergot-infected heads of rye.

droplets of a sugary solution often called “honeydew.” When disseminated to other flowers, the conidia are responsible for secondary infection.

It is generally conceded that insects are the most common agents of disseminating these secondary spores. The conidia, unlike the ascospores, are not forcibly ejected, and being borne in a sticky liquid they are subject to wind dissemination only as the wind may blow one plant's head against another. Moreover, it is a matter of common observation that insects visit the affected florets and feed upon the sugary fluid containing the spores (Fig. 99). The fungus at this stage of development has a dis-

tinctive carrionlike odor that seems to attract insects, especially flies.

Atanasoff (1920) states that as early as 1847 Leunis observed the insects visiting the infected florets and suspected that they were disseminating the spores. Mercier (1911), however, was the first to investigate the subject carefully. He showed that a fungus gnat (*Sciara thomae* L.) actively disseminated the spores both externally and internally. He showed that the spores were passed through the intestinal tract of the insect without losing their viability. The habit of flies of regurgitating the contents of the crop when feeding would appear to make them particularly effective as vectors.

Atanasoff (1920) lists more than 40 different insects that have been observed feeding upon the sugary secretion of ergot. Many of these feed not only on the honeydew containing the spores but also on the pollen of healthy flowers, a fact of considerable significance from the standpoint of inoculation efficiency. Those insects which regularly visit healthy heads as well as diseased ones would obviously be more effective vectors than those which limit their visits to diseased heads. Despite the large amount of convincing observational evidence of insect dissemination of ergot, relatively little experimental work has been done on the subject, and there are many obscure points that need further study.

Bark Beetles and Blue Stain of Coniferous Trees.—The sapwood of many species of coniferous trees is attacked by fungi that stain it dark blue; although the fungi do not decay the wood, the discoloration greatly reduces its market value (Fig. 100). Although blue stain is most frequently found in felled timber, it is not uncommon in living trees that have been weakened by drought, fire, or some other adverse factor. When living trees are attacked, the transpiration stream is interrupted, and the tree dies from the top downward. Blue stain is caused by several different fungi, but species of *Ceratostomella* or related genera are the most common ones. The genus *Ceratostomella* has been divided into two groups by von Höhnelt (1918) who applied the generic name *Linostoma* to those species with obvious affinities to *Ceratostomella pilifera* (Fr.) Wint. Because *Linostoma* had been used previously for a genus of flowering plants, Sydow (1919) substituted the name *Ophiostoma*. Goidanich (1934,

1936) divided the group still further by creating a new genus *Grosmannia*, based on the fungus described by Grosmann (1932) as *C. penicillata*. For convenience the generic name *Ceratostomella* will be retained in this discussion.

An association of insect injury with blue stain had been observed for many years, but the injury caused by insects was considered only as a weakening influence along with drought, fire, and other factors. Von Schrenk (1903) was perhaps the first to suggest that a blue-stain fungus is disseminated by beetles. He studied the association of the blue stain of western yellow pine



FIG. 100.—A section cut from a log of red pine, the sapwood of which is affected with blue stain caused by *Ceratostomella ips* and transmitted by bark beetles.

(*Pinus ponderosa* Lawson) with attacks of *Dendroctonus ponderosae* Hopk. He suspected that these beetles were disseminating the blue-stain fungus but was unsuccessful in isolating it from the insects and concluded that the spores were wind-borne and entered through the holes made by the beetles. No further study of the association was made until 1928 when Craighead called attention to the constant association of bark beetles (*Dendroctonus*) and blue stain in southern pine, pointing out that the direct injury caused by the beetles attacking living trees was not sufficient to cause the ensuing rapid death of the trees. He suggested that the blue-stain fungus was largely responsible for the death of the tree and that the fungus was probably introduced by the beetles. This hypothesis was proved

correct by Nelson and Beal (1929) and Nelson (1934) who showed that the fungus, artificially inoculated into the trees in the absence of the insects, would kill them in a relatively short time. Rumbold (1930) described and named the fungus associated with



FIG. 101.—A bark beetle (*Ips grandicollis*) caught and photographed as it emerged from a log infected with blue stain. Note the glistening white masses of spores of *Ceratostomella ips* adhering to its bristles. Histological studies have shown that the beetles are also internally contaminated and that viable spores are passed out in their excrement.

these beetles *C. ips*. Goidanich later referred it to his newly established genus *Grosmannia* [*Grosmannia ips* (Rumb.) G. Goid.]. Grossman (1930) described and named several other



FIG. 102.—*Graphium coremia* of the blue-stain fungus in the pupal chamber of *Ips pini*. A, as seen from above; B, as seen in section. Note the white sticky masses of spores borne on the tips of dark compound sporophores. The spores adhere readily to the beetles as they feed on the decayed inner bark. Approx. 10×.

blue-staining fungi and has studied in some detail their association with bark beetles in Europe.

Experimental proof of the symbiotic nature of the association of *C. ips* Rumb. and two species of bark beetles (*Ips pini* Say and

I. grandicollis Eichh.) was presented by Leach, Orr, and Christensen (1934) who studied the details of the association throughout the life history of the beetles. Freshly cut logs of Norway pine (*P. resinosa* Art.) were exposed under a variety of conditions in experiments designed for determining the degree of interdependence between the beetles and the blue-stain fungi. The ends of some

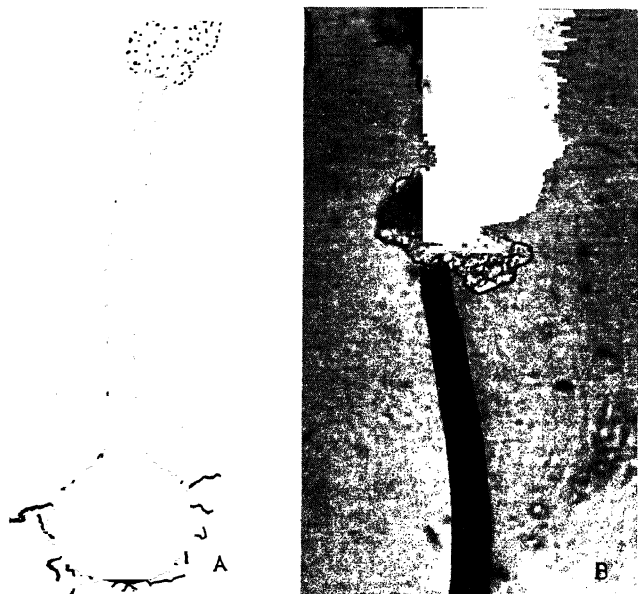


FIG. 103.—A, a perithecium of *Ceratostomella ips* (approx. 50 \times); B, the tip of a perithecium mounted in water showing the ascospores oozing from the beak in a sticky matrix. Approx. 75 \times .

of the logs were sealed, and others were left without sealing. The logs were exposed in such a way as to exclude insects from some and to exclude wind-blown spores from others. It was proved by these experiments that the blue-staining fungi, along with certain yeasts, are universally introduced by the beetles and rarely, if ever, in any other way.

The beetles, as they emerge from infested logs, are contaminated with the spores of the fungi both internally and externally (Fig. 101). Spores ingested by the beetles pass through the

intestinal tract and pass out in the excreta uninjured. As the new brood of beetles develops, the fungus grows in the sapwood and inner bark and fructifies on the walls of the tunnels and pupal chambers. Both the conidia and ascospores of the blue-staining fungi are borne in a sticky matrix in a way that ensures thorough contamination of the new brood of beetles. The conidia are borne in *Graphium* coremia in white sticky masses at the apex of intertwined, dark-colored sporophores. The coremia are often found on the walls of the pupal chambers or egg channels (Fig. 102). The ascospores are borne in black, long-beaked perithecia. In the presence of moisture, the ascospores ooze out



FIG. 104.—Perithecia of *Ceratostomella ips*, growing on the wall of a bark beetle tunnel, with their beaks pointing toward the center of the tunnel. Note the white masses of ascospores on the tips.

in white sticky masses (Fig. 103). The perithecia develop on the walls of the tunnels with the beaks pointing inward, a position well adapted for ensuring contamination of the beetles (Fig. 104). The new brood feeds in the fungus-infested inner bark for some time before emergence, a habit that is conducive to both internal and external contamination (Fig. 105).

There are usually two broods of the beetles, the first one emerging in midsummer. The freshly emerged beetles seek new trees or logs, bore into the bark, and start new broods without any intermediate feeding period. The second brood emerges in late fall and spends the winter under the duff on the forest floor. When the temperature rises in the spring, the overwintering beetles emerge, bore immediately into suitable trees, and establish new brood tunnels.

The eggs of the beetles, which are deposited in niches along the sides of the brood galleries, are internally sterile, and no special anatomical structures in the insects to ensure congenital transmission of the fungus to successive generations have been observed. The fungi apparently play no important part in the nutrition of the insect, but their presence in all probability makes the living tree a more suitable medium for beetle development by weakening the tree and reducing its water content. The fungi, by their action on the inner bark, also greatly modify the microenvironment of the developing brood of insects. The fungus obviously

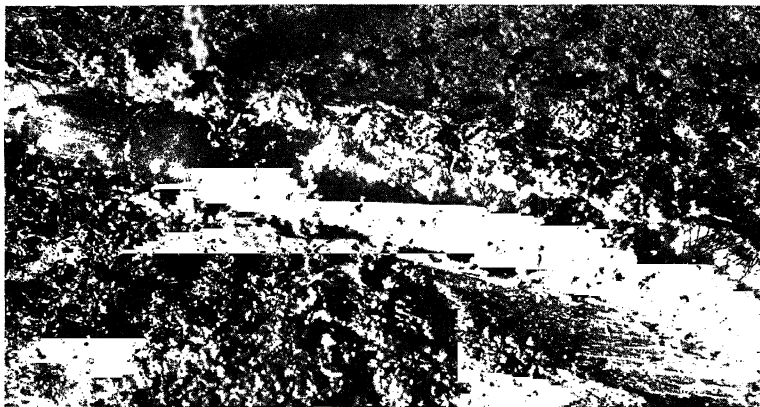


FIG. 105.—A bark beetle (*Ips grandicollis*) feeding in an old tunnel prior to emergence. Note the bristlelike perithecia of *Ceratostomella ips* along the walls of the tunnel with their tips brushing the sides of the beetle. The beetles have been observed to feed upon the perithecia. Approx. 4X.

is dependent upon the insect for dissemination and penetration into the bark of the trees. In view of these facts, the association between beetle and fungus is considered one of true mutualistic symbiosis.

In addition to *C. ips*, a previously undescribed blue-staining fungus was found associated with the bark beetles in Norway pine by Leach, Orr, and Christensen (1934). This was an imperfect fungus forming relatively large ovoid spores in white waxy stromatic masses in the pupal chambers and old egg galleries (Fig. 106). This fungus, described and named *Tuberculariella ips*, when present was usually the predominating fungus in the

brood tunnels, although *C. ips* was often associated with it. The fungus causes a blue stain of the sapwood indistinguishable from that caused by *C. ips*.

Person (1931) has shown that the yeasts associated with the beetles cause a fermentation of the sap and produce certain aromatic substances which, he thinks, may attract beetles to infested trees. This would account for the observed fact that the trees are always attacked by large numbers of beetles. Trees with a few scattered beetle infestations are extremely



FIG. 106.—Pupae of *Ips pini* surrounded by masses of spores of *Tuberculariella ips* growing on the walls of the pupal chambers. The beetles on maturity have been observed to feed on the spores many of which pass uninjured through their alimentary canals. Approx. 3X.

rare. In such isolated attacks, the beetles are usually drowned out by an excessive flow of sap and resin into the tunnels. The yeast associated with these and several other bark beetles infesting pines has been studied by Bramble and Holst (1935) and was described in detail by Holst (1936) who named it *Zygosaccharomyces pini*.

It is probable that certain species of fungi are associated in some way with all bark beetles although the extent and the economic importance of the association may vary. Rumbold (1936) has recently shown that *C. ips* is associated also with *Ips emarginatus* (Le C.), *I. integer* (Eichh.), and *I. oregoni* (Eichh.)

on several species of conifers in the Pacific Coast forests. She also has described two new species, *C. piceaperda* associated with *D. piceaperda* on spruce in eastern Canada, and *C. pseudotsugae* associated with *D. pseudotsugae* on Douglas fir and larch on the Pacific Coast. The extremely destructive Dutch elm disease is caused by an association of the several species of elm bark borers and *C. ulmi* (Schw.) Buisman. In many respects, the relationship is strikingly similar to that between the bark beetles of conifers and the blue-staining fungi.

Wright (1935) has described still another association between a bark beetle and a wood-staining fungus. *Scolytus ventralis* Le C., a bark beetle infesting white fir (*Abies concolor* Lindl. and Gord.) in California is often fatal to the trees. A brown stain associated with the tunnels was found to be caused by a species of *Trichosporium*, later proved to be pathogenic to fir trees. The fungus (named *T. symbioticum*) was found to be regularly transmitted by the beetles in a manner similar in all essential respects to that described above for the blue-staining fungi. Wright concludes that the association between fungus and insect is mutually beneficial and interprets the relationship as one of mutualistic symbiosis.

In a later report, Wright (1938) described a similar association between a brown-staining fungus [*Spicaria anomala* (Corda) Harz.] and two additional species of beetles (*Scolytus praeceps* Le C. and *S. subscaber* Le C.) affecting white fir. These two beetles commonly infest the tops and branches of the trees, respectively, whereas *S. ventralis* is found mostly in the base of the tree. *Trichosporium symbioticum* was found only in the base of the tree, and *S. anomala* was limited to the tops and branches. It was shown that the fungi would grow in any part of the tree when artificially inoculated. Therefore it was concluded that the occurrence of the two fungi in different parts of the tree was due entirely to their association with their respective vectors. Both fungi killed the cambium in advance of the beetles and caused a marked reduction in moisture content of the sapwood, a condition considered favorable for the successful establishment and maintenance of individual beetle broods.

The Dutch Elm Disease.—In 1919, a destructive disease of elm trees was discovered in Holland and described by Spierenberg (1922). In the next few years, the same disease was reported

from practically all the countries of Central and Southern Europe. It was discovered in England in 1927 (Wilson and Wilson 1928) and in the United States in 1930 (May, 1930, 1931).

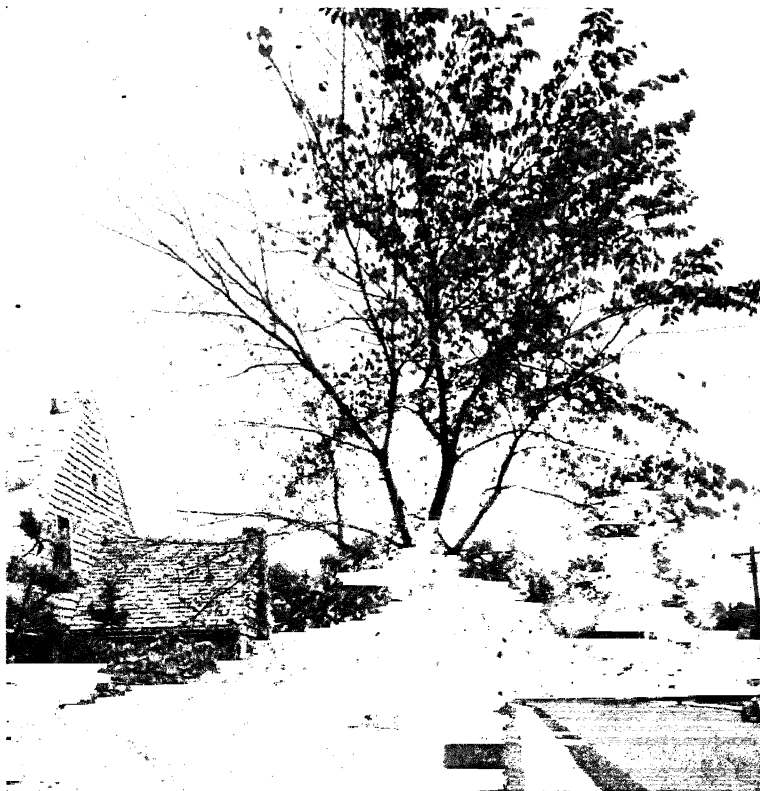


FIG. 107.—An elm tree affected with the Dutch elm disease. (*Reproduced from U. S. Department of Agriculture Circ. 322, Outbreaks of Dutch Elm Disease in the United States, by Curtis May.*)

As the disease was first recognized in Holland, it has been known generally as the "Dutch elm disease." It is extremely destructive, and practically all species of elms are susceptible. Because of the value of the elm as a shade tree, the disease has been the subject of extensive investigation in every country where it has been found.

Affected trees may be killed rapidly, dying within 1 or 2 years after infection. In other cases, the disease acts slowly and may not kill the tree for several years. The first symptoms are a wilting and drying of the leaves of terminal branches. The leaves usually drop prematurely from affected branches, only the very young terminal leaves persisting after the normal time for abscission (Fig. 107). The most characteristic diagnostic symptom, although not an infallible one, is a brown discoloration in the outer annual growth rings of the sapwood of affected branches.

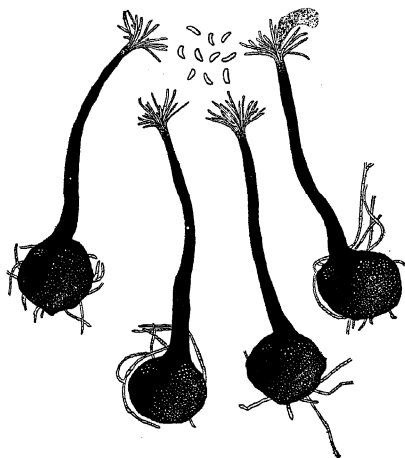


FIG. 108.—Perithecia of *Ceratostomella ulmi*. Approx. 122 X. (After Buisman.)

The discoloration may appear as a continuous or partial ring, as an arc, or as a ring made up of separate brown dots.

The Dutch elm disease is caused by a fungus, first described in the imperfect stage by Schwarz in 1922 as *Graphium ulmi*. The perfect stage was discovered and described by Buisman (1932) as *Ceratostomella ulmi* (Schw.) Buisman. It was later referred to the genus *Ophiostoma* (Nannfeldt 1932). The fungus is closely related, and very similar, to the fungi causing blue stain of coniferous trees which are disseminated by the pine bark beetles (Fig. 108).

The method of dissemination of *C. ulmi* was not known for several years after its discovery. Marchal (1927) and Wollenweber and Stapp (1928) were the first to point out the possible

significance of insects in the spread and development of the disease. These authors found the pathogen fruiting abundantly in the pupal chambers and galleries of the bark beetle (*Scolytus scolytus* Fabr.). This discovery, together with the constant association of the disease with the insect attacks and certain observations on the sporadic appearance of the disease near older infected trees, led them to the assumption that the insect was a vector of the disease. The correctness of this assumption has since been verified by numerous workers, including Betrem (1929), Fransen (1931, 1932, 1935, 1939), Fransen and Buisman (1935). In addition to *S. scolytus* Fabr. *S. multistriatus* Marsh. was soon shown to be an important vector (Fransen 1932, Collins 1935, Collins *et al.* 1936). The latter insect is the principal vector of the disease in the United States where *S. scolytus* has not yet become established. Another species of *Scolytus*, *S. sulcatus* Le C., has been found in and near the region of Dutch-elm-disease infestation and is considered as a possible vector of much importance. The biology of the beetle has been studied by Pechuman (1938) who points out that its life habits are very similar in many respects to that of *S. multistriatus*. The apple is its preferred host; but it breeds also in elm, in mountain ash, and in peach trees. Like *S. multistriatus*, it has the habit of feeding on the crotches of young twigs of vigorous trees before seeking weakened trees in which to breed and in this respect should be an effective vector of the disease.

Several other genera of insects have been incriminated in the spread of the disease or have been proved to be potentially important, but the bark beetles appear to be the principal vectors in all countries where the disease is known. In addition to the above-mentioned species of bark beetles, *S. sulcifrons* Rey., *S. affinis* Egg., *S. laevis* Chap., and *S. pygmaeus* F. are said to be important vectors in Europe (Goidanich and Goidanich 1934, Kalandra and Pfeffer 1935). Collins *et al.* (1935) have shown that the native American elm bark beetle (*Hylurgopinus rufipes* Eichh.) is also a vector, but it probably is not so effective as the species of *Scolytus*. In addition to the bark beetles, the following insects also have been mentioned as vectors, with less convincing proof: *Ptelobius vittatus* F., *P. kroatzi* Eichh., *Saperda punctata* L., *S. tridentata* Oliv., and *Magdalis armigera* Geoff.

The significance of *Saperda tridentata* as a vector has been investigated by Tyler, Parker, and Pechuman (1939). This insect is prevalent in the area of Dutch-elm-disease infestation near New York, N.Y. It breeds in the bark of diseased elms, and many adults caught after emerging from the bark were found to be contaminated with *C. ulmi*. Inasmuch as the adults feed



FIG. 109.—Life stages of the elm bark beetle (*Scolytus multistriatus*). A, eggs as they are deposited in niches along the brood tunnel; B, an adult beetle; C, a larva; D, a pupa, lateral view; E, a pupa, ventral aspect. 13×. (After Readio.)

on leaf blades, mid-ribs, petioles, and succulent elm shoots, it has abundant opportunity to transmit the disease. It was shown experimentally that the feeding wounds made by feeding adults could serve as avenues of entrance for the pathogen. Also, infection was obtained by subjecting young elms to beetles that had been artificially contaminated with spores of the fungus.

No successful inoculations with naturally contaminated beetles were reported. It is to be concluded from this work that the insect is a potential vector, but its significance in nature remains to be determined.

A comprehensive account of the disease and a review of the pertinent literature is given by Clinton and McCormick (1936). The most complete study of insect transmission and the biology of the elm bark beetles in Holland is that of Fransen, an extensive account of which was published in 1939. Good descriptions of the disease and its insect vectors in the United States have

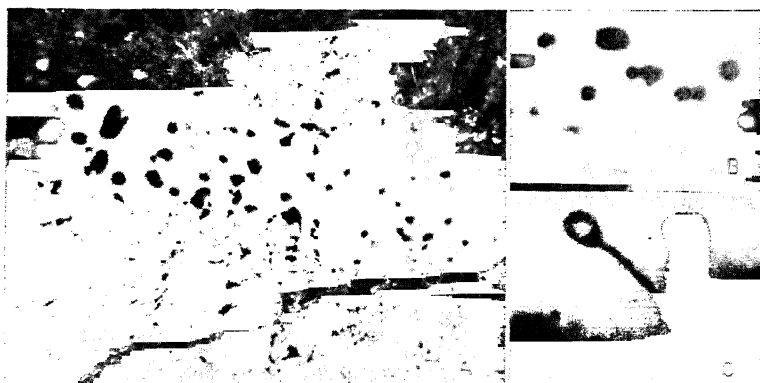


FIG. 110.—Coremia of *Ceratostomella ulmi* in the egg galleries of an elm bark beetle. The white sticky masses of spores adhere readily to the insect's body, but are not adapted to dissemination by other means. (After Clinton and McCormick.)

been published also by Welch, Herrick, and Curtis (1934), Readio (1935), and by McKenzie and Becker (1937). Collins (1938) has mapped the known distribution of *S. multistriatus*, *Hylurgopinus rufipes*, and the disease in the United States as of 1937. The former species was known to occur in three distinct centers of infestation: one in eastern Massachusetts and southern New Hampshire; one in New York, New Jersey, Connecticut, southwestern Massachusetts, Delaware, and eastern Pennsylvania; and one along the Ohio River in western Pennsylvania, southern Ohio, West Virginia, southern Indiana, and northern Kentucky. The beetle was found in five locations in eastern West Virginia and in one location just across the Potomac

in Maryland. *Hylurgopinus rufipes* is generally distributed throughout the eastern part of the United States and Canada. It probably extends throughout the range of the elm. In 1939, the disease was known to occur in a major area of infection near New York, N.Y., and in New Jersey and Connecticut and in several scattered infections in Maryland, Ohio, Indiana, and West Virginia.

The smaller European elm bark beetle (*S. multistriatus*) (Fig. 109) breeds in and under the bark of elm trees. In general, it breeds only in trees that are weakened by disease, drought,

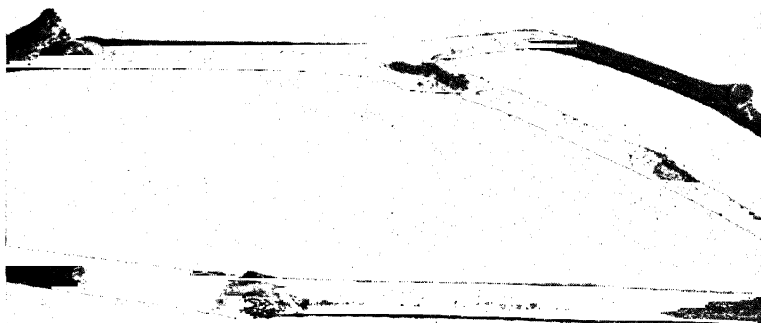


FIG. 111.—Crotch feeding wounds made by *Scolytus multistriatus* after emerging from a diseased tree and before entering a new tree to establish a brood gallery. Much of the initial infection of elm trees takes place here. (After McKenzie and Becker.)

or other unfavorable conditions. The insect survives the winter in the larval stage under the bark, and the new brood of beetles emerges in early spring. Before entering the bark of other trees to establish brood tunnels, the beetles feed for about ten days on the inner bark of the tender twigs of healthy vigorous elm trees. Numerous investigators have shown that the elm-disease pathogen fruits in the old brood and larval tunnels and that the beetles emerging from diseased trees are practically always contaminated with the spores both internally and externally. Spores borne in sticky masses on *Graphium* coremia constitute the chief source of contamination (Fig. 110). The contaminated beetles may inoculate the tree in the feeding wounds on young

branches (Fig. 111), or the fungus may be introduced by the beetles when they bore into the bark to establish brood tunnels. The brood tunnels (which are characteristic for each species of *Scolytus*) always run parallel to the grain of the wood and are 1 to 2 inches in length (Fig. 112). The eggs are laid in small niches along the sides of the tunnel (Fig. 109a). The larvae, on hatching, bore out at right angles; but, as they enlarge, their tunnels spread out so that those arising at the ends of the main tunnel may be parallel to the grain when the larvae mature (Fig. 112). The pupal chambers are formed at the end of the

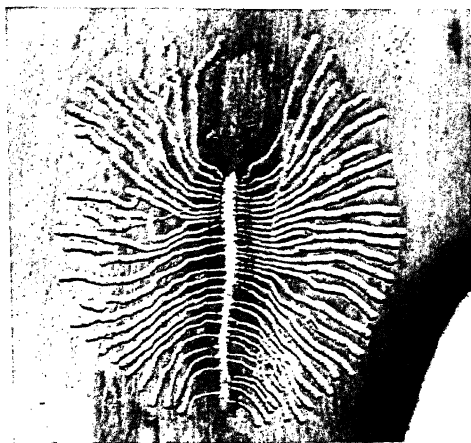


FIG. 112.—A complete brood gallery of *Scolytus multistriatus* in the bark of a diseased elm tree. (After McKenzie and Becker.)

larval tunnels, and when the adults mature they bore circular holes in the bark through which they escape. There are usually one or two broods of the beetles, the second brood serving to carry the insect over winter as larvae or pupae under the bark (Fig. 109). The larger European elm bark beetle (*S. scolytus*) according to Fransen (1939) has two to four broods per year.

The relationship between elm bark beetles and the Dutch elm disease is similar in many respects to that between the bark beetles of coniferous trees and the blue stain of conifers. The chief point of difference is in the feeding habits of the adult beetles. The pine bark beetles (*Ips. spp.*) feed for a time on

the old inner bark before emerging and do not feed on the twigs before entering a new tree to establish brood tunnels. This difference in feeding habits is of great significance in the economic importance of the two diseases. Both groups of insects require weakened trees for the establishment of brood tunnels and usually attack such trees in a gregarious manner. The pine bark beetles depend upon drought, fire injury, or other adverse factors for providing the weakened trees. The elm bark beetles, on the other hand, by their habit of feeding on the tender twigs of vigorous, healthy trees before breeding in the weaker trees, inoculate the vigorous trees with the pathogen. By the time a new brood of insects emerges from the infested trees, the vigorous trees that were inoculated by the feeding beetles have been weakened by the fungus and provide additional breeding places for the insect. Thus a vicious circle is established that makes the elm disease destructive to vigorous trees, while the blue-stain disease of pines remains largely a disease of trees that have been weakened previously by some other factor.

The native elm bark beetle (*H. rufipes*) rarely ever feeds upon the twigs of healthy trees before entering the bark of weakened ones to establish breeding tunnels, and for this reason it has been considered of relatively little importance as a vector. However, Kaston and Riggs (1938) have shown that this species usually makes a short feeding tunnel through the bark of the trunk and larger branches of healthy trees and feeds for a while on the inner bark (Fig. 113) before establishing brood tunnels. The significance of these feeding tunnels in the transmission of the disease has not yet been determined, but they are of much potential significance. Kaston (1939), who has published a complete account of the life history and habits of this insect, considers them "analogous to the well-known crotch and twig feeding injuries of *Scolytus multistriatus*, and hence important in relation to the spread of the Dutch elm disease."

D. L. Collins (1938) has studied the feeding habits of *S. multistriatus* from the viewpoint of its role as a vector. He has shown that the distribution of feeding wounds on trees in generally infested areas is very erratic. In general, the trees near infested trees and those near weakened trees that are attractive as breeding places are more subject to feeding wounds, and consequently to infection, than those not so situated.

The numerous associations between bark beetles and fungi that have been described suggest that the association is a general one, but the sudden appearance of the Dutch elm disease in western Europe where the elm bark beetles have been known for a long time seems to indicate that this particular association is not one of long standing. This view is further supported by the fact that *S. multistriatus*, an active vector of the disease, was introduced into the United States in the vicinity of Boston, Mass., prior to 1909 (Chapman 1910) and became established

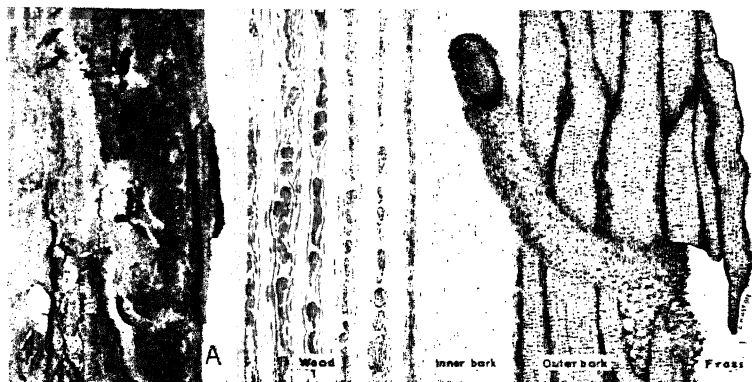


FIG. 113.—Shallow feeding tunnels made by *Hylurgopinus rufipes* in the bark of an elm tree. These tunnels are never extensive and are distinct from the breeding galleries made in weakened trees. A, a surface-view photograph; B, a diagrammatic section of a feeding tunnel. Note that the tunnel extends into the inner bark but not into the sapwood. (After Kaston and Riggs.)

there, but the disease was not introduced with it. However, when the same insect was introduced near New York, N.Y., after the disease had appeared in Europe, the disease also was brought in. These facts suggested the theory (Leach 1935) that the Dutch elm disease as a serious economic factor may have had its origin in Western Europe as a result of the association of the beetles with a new fungus which was not a serious pathogen in the absence of a suitable vector. The association of some sort of fungus with bark beetles appears to be so general that it is only reasonable to suspect that *C. ulmi*, because of its greater efficiency in providing breeding places for the beetles, is replacing a less efficient fungus symbiote. In view of this very general associa-

tion of bark beetles with fungi, it would be interesting to know what fungi, if any, are associated with the beetles in regions where the Dutch elm disease does not occur.

The discovery of Walter (1937) that there are nonpathogenic strains of *C. (Graphium) ulmi* suggests the possibility that the pathogenic strains may have arisen by mutation from a previously harmless fungus symbiote of the beetle. If this were true, the nonpathogenic strains might be found associated with the beetles in the disease-free areas. In so far as the author knows, no study of the fungi associated with the beetles in disease-free areas has been published.

The possible role of mites as vectors of the Dutch elm disease has been discussed by Jacot (1934, 1936). Tyroglyphid mites, which are themselves transported in the hypopial stage from tree to tree by insects that breed in the bark of infected trees, are potential vectors, but their importance in this respect was not determined by Jacot. Fransen (1939), also, has described the association of mites (*Pseudotarsonemoides innumerabilis* Vitzth.) with the elm bark beetles and *C. ulmi*. According to Fransen, the mites transport the spores of the fungus from the egg galleries to the walls of the pupal chambers and, by their activity, stimulate the fungus to sporulate more profusely than it does in the absence of the mites. For a more complete discussion of mites as vectors of this and other diseases, see Chap. XI.

The association of a destructive plant disease such as this one with insect vectors adds much to the complexity of the problem of control. Attempts are being made to eradicate the Dutch elm disease in the United States. In Holland and other European countries, earnest efforts are being made to protect the remaining elms by controlling the beetles and protecting the elms against beetle attack. The success of these efforts depends upon many factors, one of the most important of which is a thorough knowledge of the pathogen, its vectors, and the intricate association of the two. Because of the economic and aesthetic value of the elm, extensive and thorough investigation of the disease is imperative.

Fig Diseases.—In California, the fig is affected by a group of diseases commonly referred to collectively as "fruit spoilage diseases." These diseases are largely dependent upon insects for their spread and development. They are destructive and

often cause enormous losses. The three most common diseases are *endosepsis*, *smut*, and *souring*.

Endosepsis, an internal rot of the fig fruit, was first recognized and described by Caldis (1925, 1927), who studied it in California from 1922 to 1925. The disease was prevalent and destructive in 1927 and has been troublesome since then. *Endosepsis* affects only the caprifig, and the fig wasp (*Blastophaga psenes* L.) (Fig. 114) is the principal vector of the fungus pathogen (*Fusarium moniliforme* var. *fici*).

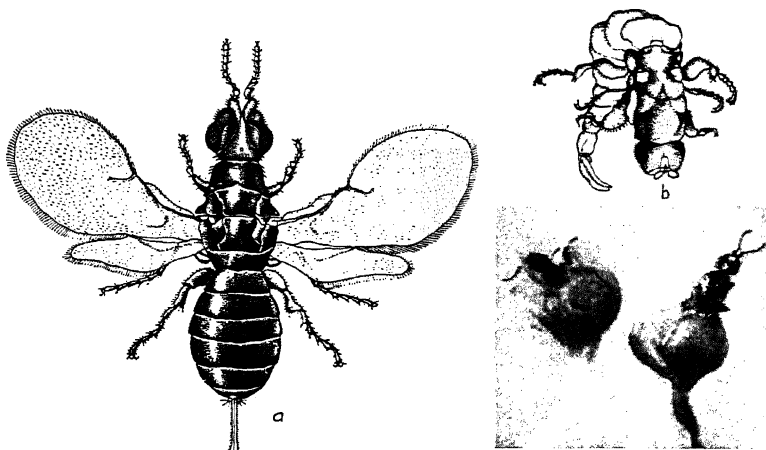


FIG. 114.—The fig wasp (*Blastophaga psenes*). a, female; b, male; c, a male and female emerging from galls from a caprifig. The female is impregnated by the male before she emerges from the gall. On leaving the fruit the female becomes dusted with pollen from the staminate flowers that surround the opening. If the fruit is diseased the insect may also become contaminated with spores and transport them to healthy fruits of the edible fig. (After Smith and Hansen.)

In order to understand the relation of the insect to the disease, it is necessary to know the life history of the insect and its relationship to the development of the fig fruit. This relationship is briefly and clearly presented by Smith and Hansen (1931). The fig "fruit" is in reality an aggregation of fruits borne in the cavity of a hollow receptacle, known botanically as a "synconium" (Fig. 115). The caprifig of California is of the *Smyrna* type, and is a dioecious, insect-pollinated plant. The synconium that becomes the edible fruit bears no pistillate flowers, whereas the caprifig has both male and female. The caprifig has three crops

of fruit each year. The first or spring crop is called the "profichi," the second or summer crop the "mammoni," and the third or fall crop the "mamme."

The *Blastophaga* insects live over winter in the galls or enlarged florets of the mamme crop. In early spring, the male *Blastophaga* emerge from the galls and fertilize the females while the latter are still in the galls (Fig. 114). After the females have been fertilized, they emerge, leave the old mamme fruits, and enter the

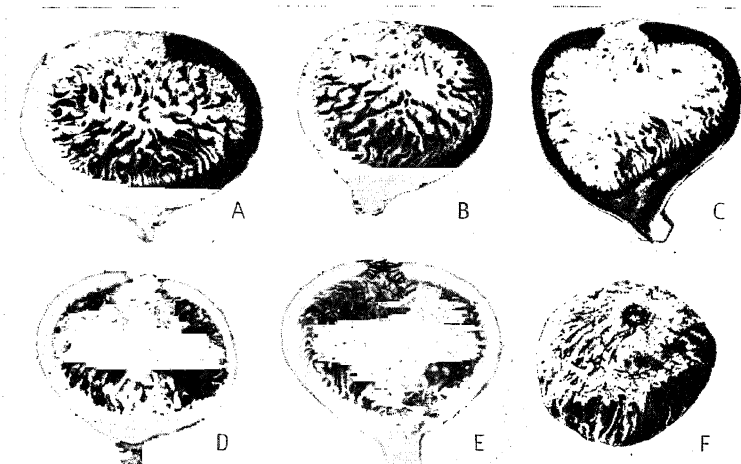


FIG. 115.—Endosepsis of *Calimyrna* figs. A, a sound fig fruit consisting of a hollow fleshy receptacle with an aggregation of flowers or fruit arising from its inner wall; B, an early stage of endosepsis with only the flowers around the "eye" affected; C-E, more advanced stages of endosepsis; F, external view of a badly diseased fruit. (After Smith and Hansen.)

young developing profichi crop. Here they oviposit in the ovules of the young flowers. The eggs hatch, and the larvae develop and pupate in the galls formed from these flowers. This new brood of insects is ready to emerge in early summer when the crop of figs on the edible-fig trees is developing. When the female insects leave the profichi figs, they rub against the staminate flowers which surround the opening in the fruit and become covered with pollen (see Fig. 24). The insects carry the pollen into the undeveloped edible fruit and pollinate the flowers while seeking a place to oviposit. Oviposition, however, is not effected,

because the styles of the flowers in the edible fig are too long. The flowers, however, are successfully pollinated, and the fruits develop normally. Some of the insects enter the third, or mamme, crop of figs where they pass the winter as larvae or pupae in the galls. Various kinds of special manipulation are practiced by growers to ensure caprification of the crop of edible figs. Usually the profichi crop is collected when the females are ready to emerge, and the figs are hung in wire baskets in the branches of the edible-fig trees.

Endosepsis is a firm dry rot of the fig fruit (Fig. 115). It is not easily detected from the exterior until the fruit begins to ripen. The affected tissues are water-soaked at first, becoming brown with a tinge of pink color. In later stages, the fruit is dry, seedy, and tasteless. There is no fermentation or souring of the tissues associated with endosepsis.

Caldis has shown that the spores of the pathogen are introduced into the edible fig by the *Blastophaga* insect. The fungus infects and sporulates also in the tissues of mamme and profichi fruits. The insects are usually contaminated with the spores as they emerge from the infected fruits. The fungus is not associated with the eggs, larvae, or pupae. The adults are contaminated only after they emerge from the galls. Caldis proved experimentally that figs pollinated by insects from noninfected caprifigs remain healthy, but when pollinated by insects that come from infected caprifigs the fruit becomes diseased with endosepsis. Two species of bacteria are constantly associated with the fungus and are also disseminated by the insect, but neither species appears to be pathogenic. The fungus (*Fusarium moniliforme* var. *fici*), however, is pathogenic when used in pure culture as inoculum.

Hansen (1927, 1928) demonstrated that caprifigs could be successfully disinfected without injury to the insects by washing the interior of the fruit with a 0.2 per cent solution of Semesan while the insects were still in the galls in the larval or pupal stages. This treatment appeared to be practical when applied to the mamme crop so that the profichi crop would be infested with noncontaminated insects.

Some success in the control of endosepsis has been obtained in the use of the treatment by the growers. In 1928, an effort was made to use the method on a large scale under state supervision.

More than 60 tons of the mamme crop was collected and sent to a central station where it was treated and incubated until the *Blastophaga* emerged to be caught in glass tubes and returned to the orchard for caprification of the profichi crop. The effort appears to have been somewhat premature for such large-scale operation, and although there was some evidence of control of the disease, the project was not entirely successful. The disease-control program resulted in a scarcity of insects for pollination, and there was a very poor set of fruit. The loss from the short

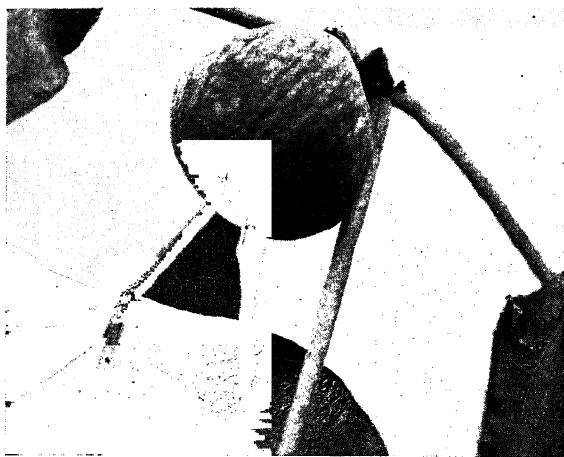


FIG. 116.—A fig fruit affected with "souring." Note the exudate from the eye.
(After Smith and Hansen.)

crop offset the gain through disease control. With more experience and better technique, the control through disinfection of the mamme crop may prove practical.

Souring is a term applied to the fermentation of the sugary sap of figs by two or more species of yeast and the resulting decomposition and contamination by bacteria and molds. Souring affects both the common parthenocarpic fig and the caprifig, but it is most typically a disease of the former which, because of its independence of the fig wasp, is not affected by endosepsis. The disease is often very destructive, sometimes causing a loss of more than 50 per cent of the crop.

Souring is a disease of the ripe fruit. The normally pink flesh loses its color and becomes water-soaked and translucent. Odors characteristic of alcoholic fermentation followed by that of acetic acid are characteristic. Gas bubbles form in the pulp, and a jellylike exudate flows from the "eye" (Fig. 116). A white scum of yeast is often evident on the surface of the affected tissue. When the fruits dry before destruction is complete, the base of the fruit dries to a black shriveled mass to which the term "black neck" is often applied. Small cankers are formed on the stem at the point of attachment if the fruit does not drop.

The work of Smith and Hansen (1927), Caldis (1930), and Hansen (1929) has shown that the yeasts responsible for souring, as well as the secondary fungi that follow, are introduced largely, if not entirely, by insects. The most important of the vectors of souring are the dried-fruit beetle (*Carpophilus hemipterus* L.) (Fig. 117) and the fruit fly (*Drosophila ampelophila* Leow.). Both these insects feed on, and breed in, decaying citrus fruits, melons, etc., where they have ample opportunity to become contaminated with yeast. When the "eye" of the fig is open, these insects enter in search of food and carry the yeast and fungus spores with them. The disease has been produced by caging the dried-fruit beetle with healthy fruits, and fruits have been protected from the disease by excluding the insects. Caldis (1930) has shown that the yeasts are transported both externally and internally by the dried-fruit beetle.

Hansen and Davey (1932) have demonstrated that several species of thrips and mites enter young fruits even before the eyes are "open" and that they introduce spores of several different species of mold as well as a few yeasts in about 15 per cent of the cases. Unsuccessful attempts to introduce spores into the interior of the figs by forcibly blowing them against the eye with an atomizer strengthen the conclusion that insects are the chief agents of inoculation and that wind is of little importance. Because the dried-fruit beetle as well as the fruit flies breed in decaying fruits, the destruction of any waste fruits that might serve as breeding places is recommended as the most important control measure.

Smut of figs is not a true smut but a mold, being caused by a strain of *Aspergillus niger*. It is one of the oldest and best known

diseases of the fig. It has been known in Southern Europe for many years and is mentioned in the earliest records of the fig industry. All kinds of fig are affected, but smut is most common on caprifigs.

In the earlier stages of smut, small, brown, soft spots are formed on the interior flesh. In later stages, when the fruit is ripe or even after drying, masses of black spores are formed. Through the work of Hodgson (1918) and Phillips, Smith, and Smith (1925), it has been proved that the spores of *Aspergillus*



FIG. 117.—Dried-fruit beetle (*Carpophilus hemipterus*) emerging from a decaying orange fruit. Such insects are thoroughly contaminated with yeasts, molds, and other microbes. When they enter the fig fruit, diseases such as souring or smut usually follow. (After Smith and Hansen.)

are introduced into the figs chiefly by the dried-fruit beetle (*C. hemipterus* L.) (Fig. 117) and to a lesser extent by *D. ampelophila* Leow. According to Hansen and Davey (1932), thrips and mites enter the very young fruits, and it has been shown that they may introduce spores of *Aspergillus* as well as other fungi.

The control of smut, like the control of souring, depends upon reducing the number of contaminated beetles and thrips that reach the fig fruits. The destruction of waste fruits that serve as breeding places for the beetles and the fungi is recommended.

Perennial Canker of Apple and the Woolly Aphis.—In 1925, Zeller and Childs described a disease of apples in Oregon caused by a fungus, which they named *Gloeosporium perennans*. The

disease causes both a rot of the fruit and a canker of the branches. The cankers are characterized by successively formed concentric rings of callus growth (Fig. 118). The fungus grows actively in the cankers during the late winter months, when the callus formed the previous season is invaded and killed. In the summer, the pathogen is inactive, and the canker heals, forming a new ring of callus which in turn is killed by the fungus the following winter. This progressive, year-to-year expansion of the canker led Zeller

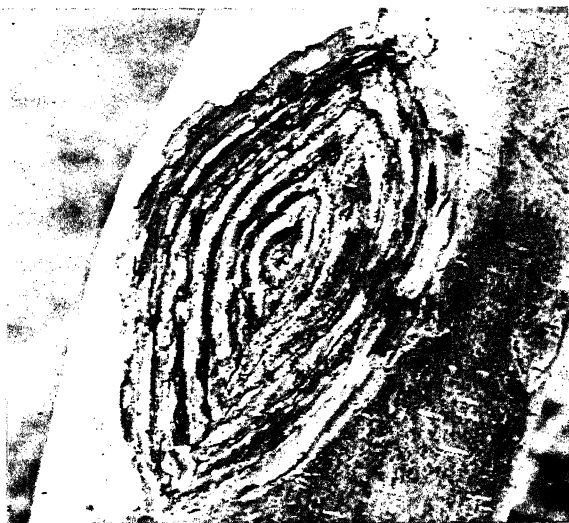


FIG. 118.—A large perennial canker of apple showing the numerous annually formed callus rings. After each year's growth of callus the pathogen is completely walled off and reinfection is dependent upon the feeding wounds of the woolly aphids. (After McLarty.)

and Childs to name the disease "perennial canker." Childs (1929) and McLarty (1933) demonstrated that in spite of the perennial character of the canker the infection is really annual in nature. They showed that in summer, when a new callus was being formed, the pathogen was completely shut out of the living tissues so that a new infection was necessary for the further expansion of the canker. Since the fungus may live for several years as a saprophyte in the dead bark and may sporulate abundantly, it is universally present in old cankers but is unable to reinfect

without the aid of some wounding agency. McLarty (1933) has shown that the necessary wounds are provided by the wooly aphid (*Schizoneura lanigera* Hausmann) which commonly feeds on the newly formed callus tissue (Fig. 119). Small blisterlike galls of delicate tissue are formed where the aphids feed. Upon freezing, these galls rupture and provide the wounds through which the fungus reinfects.

The original, or primary, infection of the branches occurs chiefly through pruning wounds and usually follows aphid infestation of the callus formed on the wound. Small "button cankers"



FIG. 119.—Two young perennial cankers showing the wooly aphid feeding on the tender callus tissue. (After Childs.)

are often produced as a result of direct infection through the lenticels; but these are very superficial and, because they are not attractive to the aphids, usually persist for only 1 year and then heal over completely. There is no experimental evidence to show that the aphids disseminate the fungus, but it is believed by many observers that they do so to some extent.

The life history of the wooly aphid is somewhat complicated and is not completely known. It can live on both the roots and the aboveground parts of fruit trees as well as on the foliage of the elm, where the winged forms and the egg-laying generation occur. Eggs are deposited in crevices of the elm bark where they survive the winter and hatch in the spring. The insects may also survive the winter as adults in the cankers on apple

trees. Small swellings are caused on the roots of the apple trees, but these are soon destroyed by decay. The cause of this decay of infested roots has never been determined.

The association of the woolly aphis with this disease is not entirely fortuitous, for there is a measure of mutualistic symbiosis involved. The crevices and old bark of the cankers provide protection for the aphids against predators, parasites, and other unfavorable factors of the environment (Fig. 120). In turn, the aphids provide the wounds necessary for reinfection, and



FIG. 120.—A, perennial cankers after 1 year's growth, still covered with the dead bark; B, the same cankers with dead bark removed to show the woolly aphids to which the canker affords both food and shelter. (After Childs.)

in the expansion of the canker and the resulting callus additional food and protection for the aphids are provided. Thus the insect and the fungus are mutually benefited by the association.

A knowledge and appreciation of the part played by the aphids in the development of the cankers have helped in the control of the disease. Control of the canker has been accomplished largely through the control of the aphids. More rigid inspection of nursery stock for aphid infection is practiced, and aphids are controlled in the orchard by spraying and by cleaning out the old cankers, so that the protection for aphids will be reduced to a minimum. Cankers formed by stub pruning offer less protec-

tion for the aphids than those formed by close pruning, and stub pruning therefore is recommended. In Oregon, when cankers are cut out later than February, no extension of the canker takes place; for the incipient infection is removed, and new, vigorous, uninjured callus growth follows. It has been observed that the Northern Spy variety is resistant to the aphid and is also relatively free from perennial canker.

European Canker and the Woolly Aphis.—Although the details of the association have not been worked out so thoroughly as they have with respect to perennial canker, evidently a similar relationship exists between the European canker and the woolly aphis. European canker caused by *Nectria galligena* is essentially the same type of canker, the pathogen becoming active in late winter and the canker healing in summer. The close association of these insects with the European canker has been observed for a long time, but their importance in the development of the canker has not been extensively studied. Wiltshire (1915), however, described the galls made by the aphids when feeding on the callus in the cankers and concluded that infection probably occurred through the wounds made by the collapse of the galls, rather than through the earlier feeding punctures. He also noted that cankers usually occur in the year following that of heavy aphid infestation. Cotton (1918) discussed the probable relationship of aphids to the canker as follows:

Probably one of the most frequent methods by which the fungus gains admission is through injuries caused by the woolly aphis. The soft, swollen tissue produced by the aphids is very apt to become cracked during the winter, and it is probable that when the aphis returns the following season to the wounded area, *Nectria* spores are attached to their bodies and introduced into the cracks with the aphis.

Cunningham (1923) has reported in New Zealand a canker of apple caused by *Diplodia griffoni*. He concluded that infection is effected chiefly through wounds made by the woolly aphis and that the fungus is also disseminated to a great extent by the insect. The relationship between insect and fungus is probably very similar to that described for perennial canker.

Bees and the Downy Mildew of Lima Beans.—Sturgis (1898) has shown that bees are the principal agents in the dissemination of downy mildew of lima beans, caused by *Phytophthora phaseoli*

Thaxter. The disease, which was first described by Thaxter in 1889, was reported as very destructive in wet seasons in Connecticut. Sturgis (1898), following a severe outbreak of the disease in 1897, reported that one of the most serious aspects of the disease was the infection of the young ovary before the fall of the blossoms. Careful observations led him to conclude that bees, which normally pollinated the flowers, were largely responsible for this floral inoculation. He noted that the young pods became infected only at the base of the ovary and on the



FIG. 121.—Bean blossoms showing the method of inoculation with downy mildew (*Phytophthora phaseoli*) through the agency of bees. A, a normal flower with all parts in position before pollination; B, a flower with keel depressed and style protruded after it has been visited by a bee; C, a sectional view of the flower with petals removed to show ovary; D, ovary from an infected flower showing mildew growing and fruiting on the style and at the base of the ovary, the two points of contact with the insect. (After Sturgis.)

style and that these were the two points where the bee in search of nectar touched the more moist and delicate tissues of the flower.

A good visualization of this relationship can be had by an examination of the flower parts shown in Fig. 121. When a bee alights upon the flower, its weight deflects both the wings (B) and keel (C) and causes the style to protrude from the keel and to touch the bee. In seeking the pollen, the bee's head brushes the base of the ovary. This is an adaptative mechanism that ensures pollination, but it also facilitates the inoculation of the young pod with the spores of *Phytophthora phaseoli*. The

style and the base of the ovary are fully protected by the petals until exposed by the bees in this way, but it is at exactly these points that the young pods are infected by the fungus.

Plant Bugs and Stigmatomycosis.—A characteristic type of injury to plant tissues, associated with the feeding punctures of many species of plant bug, has been recognized for many years. The injury generally has been attributed to the toxic effect of the salivary secretions of the insect; but more recent investigations have shown that, in many cases, it is caused by microorganisms introduced into the plant tissues by the insects while feeding.

Nowell (1917a, 1917b, 1918) apparently was the first to point out the true nature of the injury associated with the feeding punctures of certain of these insects. He investigated the internal diseases of cotton bolls in the West Indies and showed that the staining of the lint was caused by four yeastlike fungi, the most important of which were two species of *Nematospora*. These fungi were found abundantly internally, in immature unopened bolls on which there was no visible surface infection. The disease was produced experimentally by subjecting healthy bolls to the feeding of two genera of plant bug, the cotton stainers (*Dysdercus spp.*) and the green bug (*Nezara viridula* L.). Two other insects [*Leptoglossus bolteatus* and *Phthia picta* (Drury)] were incriminated but were considered of less importance than the first two. No other natural method of inoculation was found. When very young bolls are fed upon, they drop off; older ones remain attached, but the lint is stained. Stringent efforts to control the bugs resulted in fair control of the disease.

The nature of the association of the fungi with the beetles was not determined by Nowell. There is some evidence that there are some individual bugs which do not introduce the fungi when feeding. A slight proliferation of the tissues about the point of puncture is the only visible effect of the feeding of such insects. The way in which the fungi survive the winter, the source of contamination of the insects, and the extent of the association in the various stages of the life history of the insects were not determined.

The life history and habits of the principal plant bugs that are injurious to cotton bolls have been described by Morrill (1910). The cotton stainers oviposit in the soil, and the eggs hatch in

about 8 days. There are five nymphal instars, all of which are capable of transmitting the fungi. From 45 to 50 days is required for the completion of the cycle.

The fungi of stigmatomycosis were studied in more detail by Ashby and Nowell (1926), who identified four species (*Spermophthora gossypii* A. and N., *Eremothecium cymbalariae* Borzi, *Nematospora gossypii* A. and N., and *N. coryli* Peglion). Of these, *N. gossypii* and *N. coryli* were most commonly associated with the disease of cotton bolls. These workers were the first to apply the term "stigmatomycosis" to the disease.

The work of Nowell, who demonstrated that the disease was caused by fungi transmitted by plant bugs, was soon verified and extended by several workers in different parts of the world. Laycock (1925) demonstrated the association of *D. supersticiosus* and other species of plant bug with *N. gossypii* in the production of the disease in Nigeria. Rhind (1927) found the disease in Burma, caused by *N. gossypii* and *N. coryli* to be disseminated by *D. cingulatus* A. Hansford (1930) proved that *N. gossypii* was disseminated and introduced into cotton bolls by *Dysdercus* spp. in Uganda and pointed out that the young nymphs were not able to transmit the fungi into the bolls. Moore (1930) reported similar relationships in South Africa between *N. gossypii* and *N. coryli* and *D. fasciatus* and *D. nigrofasciatus*. The fungi were found also in spots on several kinds of beans as well as other plants fed upon by the insects. Steyaert (1934) described essentially the same relationship in the Belgian Congo, and Pearson (1934) reported extensive studies in the Transvaal and added *D. intermedius* as a vector. Important contributions to the study of stigmatomycosis in South Africa have been made by Ullyett (1930).

The relationship of fungi to the injury to cotton associated with the feeding of Hemipterous insects has received very little attention in the United States. Cassidy and Barber (1939), however, have called attention to the injury caused to cotton by these insects in Arizona where they estimate an average loss of five dollars per bale. Fifteen different species of Hemiptera were found to feed and breed on cotton, but most of the injury was caused by eight species, the most important of which were three Pentatomidae, namely, *Euschistus impictiventris* Stal., *Chlorochroa sayi* Stal., and *Thyanta custator* (F.). In discussing

the nature of the injury these authors state, "The most conspicuous injury, and the damage most keenly realized by cotton growers, consists of the puncturing of the bolls, followed by the development of severe lint staining by introduced pathogenic organisms."

Wingard (1922) described from Virginia a "yeast spot" of lima beans and cowpeas caused by *N. phaseoli* Wingard and in

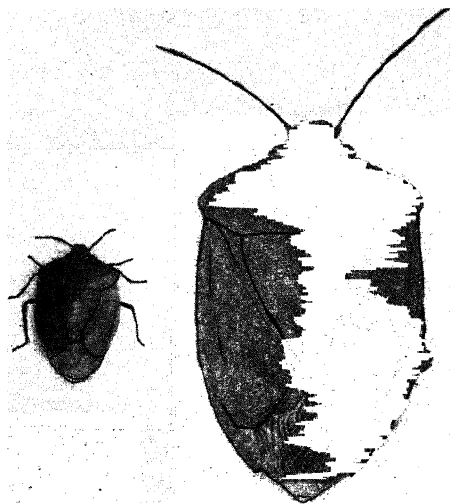


FIG. 122.—The green stinkbug (*Nezara hiliaris* Say), the vector of stigmatomycosis or yeast spot of lima beans; natural size and approx. 4×. (After Underhill.)

1925 reported proof of its dissemination by the green stinkbug (*Nezara hiliaris* Say) (Fig. 122). The bugs feed upon the pods, their beaks penetrating the young seed. Brown, sunken, necrotic lesions are formed on the seed (Fig. 123). Usually only the seeds are affected; but when the bugs feed on very young pods the necrosis is more general, and the entire pod shrivels and falls off (Fig. 124). The life history of the bug and a good description of the injury caused are given by Underhill (1934). Wingard failed to isolate the pathogen from the bug but claimed that failure was due to faulty technique. No

study was made of the source of contamination of the bugs or the method of survival of the fungus over winter.

The bugs hibernate as adults under leaves and grass on the ground. They become active in late June and begin oviposition. Eggs are deposited on the surface of leaves, fruits, or limbs of certain preferred wild hosts, especially American elder (*Sambucus canadensis* L.), black locust (*Robinia pseudoacacia* L.), and honey locust (*Gleditsia triacanthus* L.). Oviposition may continue throughout the summer, reaching a peak in the middle of July. The eggs hatch in about eight days. There are five

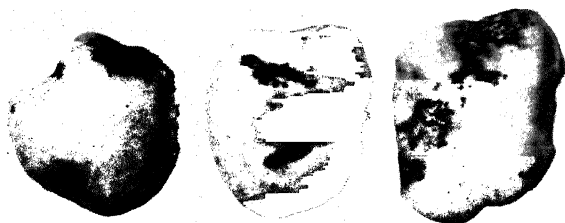


FIG. 123.—Three lima-bean seeds showing the brown necrotic lesions caused by the yeast (*Nematospora phaseoli*) inoculated through the pods by the green stinkbug (*Nezara hilaris*). (After Underhill.)

nymphal instars requiring about five weeks for complete development. There is only one brood each season. The disease apparently may be transmitted by any stage of the insect although the information on this point is not complete.

Beans are not the preferred host of the insects; they usually migrate to beans from the preferred wild hosts. The black locust, red bud, and linden trees are the most important in this respect, and it is recommended that beans should not be planted less than 100 yards distant from these trees. If practical, such trees should be removed.

Lee (1924) and Fawcett (1929) have described a disease of citrus fruits caused by *N. coryli* which they call "insipissosis" or "dry rot." Fawcett (1929) showed that the pathogen was transmitted from pomegranates to citrus fruits by the leaf-footed plant bug [*Leptoglossum zonatus* (Dall.)]. The disease

was experimentally transmitted by placing the insects, taken from infected pomegranates, on sound oranges and lemons in jars in the laboratory. The fungus was readily isolated from fruits inoculated by the bugs in this way. Attempts to isolate the fungus from the beaks of several individual bugs were not successful. Weber (1933a) has reported *N. coryli* and *N. gossypii* from citrus and several other different fruits in Florida.

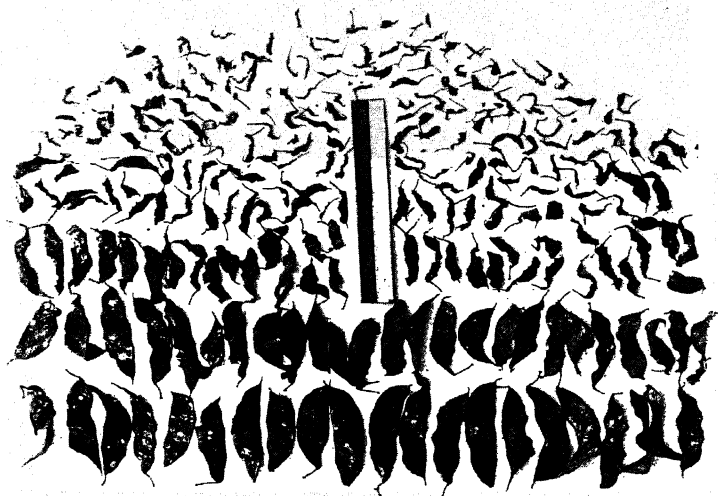


FIG. 124.—Lima-bean pods shed from a single vine as a result of yeast transmitted by the green stinkbug. (After Underhill.)

Stigmatomycosis, in some form, probably occurs on all plants on which the vectors feed.

Weber (1933b) has described a kernel spot of pecan caused by *N. coryli* and has observed its association with the feeding punctures of *Nezara viridula*. This was later verified by Demaree (1922) who suggested that "the pathological result may be caused by the mechanical rupturing of the host cells by the sucking up of the plant juices, by the injection of toxic substances into the tissues, or by the combined result of all three types of injury." In view of the described associations between the plant bugs and *Nematospora* throughout the world, it is quite probable that

kernel spot of pecans is a stigmatomycosis, in all respects similar to that on other plants and that *Nezara viridula* is the principal vector. Complete experimental proof, however, has never been established.

Wallace (1932) described a similar disease on the coffee bean which he showed was caused by *N. gossypii* and *N. coryli*. The fungi in this case were transmitted by the bug *Antestia lineaticollis*.

When one considers the injuries caused on many cultivated plants by plant bugs of this type, it seems probable that much of the injury may be due to fungi introduced by the bugs while feeding. The universal association of species of *Nematospora* with the insects in such widespread regions of the world would suggest that the association is more than a casual one. A thorough study of the possible symbiotic relationships between insect and fungus should be made. It is significant that no satisfactory explanation of the method of survival of the fungi over winter has been offered by any workers on the disease. The question of whether the fungus is constantly associated with the insects throughout their life history or whether they pick it up from some unknown source has never been answered. The problem of stigmatomycosis is in need of much more investigation.

The Anther Smut of Pinks.—The better known smut diseases affect members of the grass family, all of which are wind-pollinated. Those smuts which infect grasses through the floral organs usually form spores at flowering time which, like the pollen grains, are dependent upon wind for their dissemination. There are a few smuts that infect through the floral organs of insect-pollinated plants. In these cases, the smut spores are specially adapted to insect transmission. The anther smut of pinks (*Caryophyllaceae*), caused by *Ustilago violaceae*, is the best known example of the insect-transmitted smuts. The phenomena of infection in *U. violaceae* have been described by Brefeld and Falck (1905).

The spores of the fungus are formed in the anthers of infected plants, where they replace the normal pollen grains. The fungus infection is systemic; and although all flowers on an infected plant are usually smutted, the spores are formed only in the anthers. The spores are approximately the same size as the pollen grains, but darker in color. They are, like the pollen grains, somewhat

sticky and are not easily scattered by the wind. The flowers of the host plant open in the evening and remain open after dark. Pollination is brought about by several species of sphinx moth that visit the flowers in search of nectar. When an infected flower is visited, the insect becomes contaminated with the smut spores, which it transmits eventually to the stigmatic surface of a healthy flower. The spores find the stigmatic secretions a favorable medium for germination and growth. The infecting mycelium penetrates the young ovary but does not destroy it. When the seeds from such infected flowers are planted, they produce systemically infected plants, all the flowers of which produce only smutted anthers.

This disease is a striking illustration of the adaptation of a fungus to insect transmission. The association is probably a very old one and may have evolved along with the host and its adaptation for insect pollination.

Blossom Blight of Red Clover.—This disease, caused by *Botrytis anthophila* Bond., was first observed in Russia by Bondarzew (1913). It has been observed more recently in Wales and has been described in some detail by Silow (1933). The fungus fruits on the anthers of affected plants where the yellow pollen grains are destroyed and replaced by gray spores of the fungus (Fig. 125). Usually, all the florets on the clover head are affected, but occasionally some normal anthers are produced. The vigor of the plant is not impaired, the blighted anthers constituting the only visible symptoms. The greatest economic importance of the disease results from the decreased yield of seed.

Bondarzew (1914) observed bees visiting infected florets and suggested that they were vectors of the disease. Similar observations were made by Silow, who demonstrated experimentally that bees were the principal vectors. Red clover is largely dependent upon bees for pollination, and the bees, in searching for nectar in infected flowers, become contaminated with spores, transmitting them to the stigmatic surfaces of healthy flowers. The spores germinate and penetrate the style (Fig. 125c), the mycelium eventually reaching the ovary. The ovary is not destroyed but develops normally. The mycelium, however, persists under the seed coat (Fig. 125d), where it remains dormant until the seed sprouts. When the seed germinates, the mycelium begins to grow and systemically infects the young plant. The

growth of the fungus keeps pace with the plant; and when flowers are produced on the new plant, the mycelium penetrates the anthers and sporulates (Figs. 125*a* and *b*).

The transmission of anther blight by bees closely parallels the transmission of anther smut of pinks by the sphinx moth, and both associations resemble very much the better known phenomena of insect pollination. The life habits and methods of fructification of both pathogens are obviously adaptations for insect transmission.



FIG. 125.—*Botrytis anthophila*. *a*, a blighted anther on which the fungus is fruiting; *b*, an enlarged sporophore showing how the spores are borne; *c*, a stigma of a clover blossom showing pollen grains and *Botrytis* spores germinating and penetrating the style; *d*, a section through the seed coat of a clover seed showing the mycelium of the fungus. (After Silow.)

Tree-cricket Canker of Apple.—Parrot, Gloyer, and Fulton (1915) and Gloyer and Fulton (1916) described a canker of apple trees that was associated with the oviposition punctures of tree crickets (Fig. 126). They showed by experiments that the cankers were caused by *Leptosphaeria coniothyrium* (Fekl.) Sacc. and that the disease was transmitted by tree crickets. They called the canker "tree-cricket canker." The fungus causes also a cane blight of raspberry that is often transmitted by the same insects. In the course of the study of transmission of this

canker disease, it was found that the tree crickets disseminate other species of fungi and that they are probably concerned in the spread of several diseases.



FIG. 126.—A typical tree-cricket canker on an apple branch. The light spot in the center is the oviposition puncture where infection took place. (After Gloyer and Fulton.)

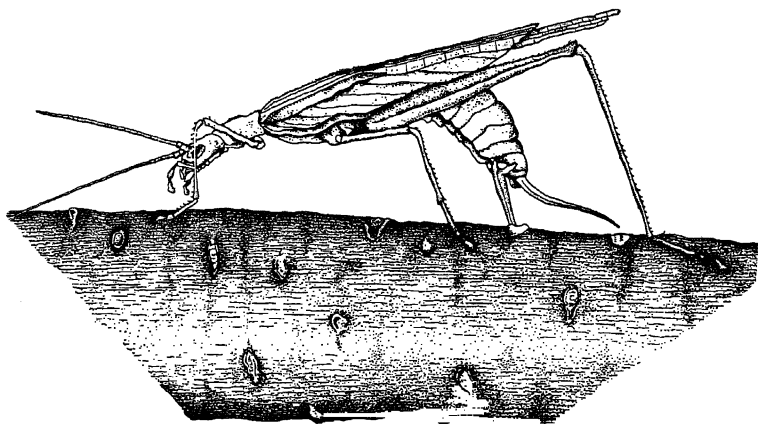


FIG. 127.—A tree cricket (*Oecanthus niveus*) in the act of oviposition. (Redrawn after Parrot and Fulton.)

In order to understand clearly how the tree crickets transmit the disease, it is necessary to know something of the life history and habits of the insects. A good description is given by Parrot

and Fulton (1914). The role of two species as vectors was studied extensively by Gloyer and Fulton (1916). These were *Oecanthus niveus* De G., the snowy tree cricket, and *O. angustipennis* Fitch, the narrow-winged tree cricket, the most prevalent species on apples in New York. In late summer, these insects oviposit in branches and twigs of apple trees. *O. niveus* oviposits chiefly in branches 1 to 2 inches in diameter while *O. angustipennis* selects smaller branches ranging from $\frac{1}{3}$ to $\frac{1}{2}$ inch in diameter. The female first chews a hole in the outer bark, then inserts her ovipositor, deepens the hole, and deposits an egg (Fig.

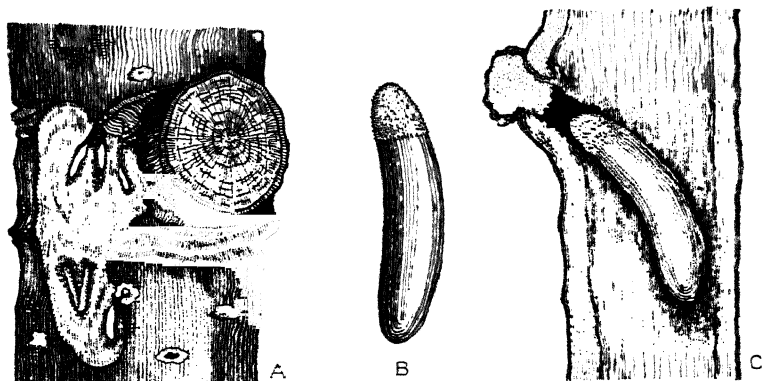


FIG. 128.—The method of oviposition of the snowy tree cricket. A, an apple twig with a portion of the bark removed to show egg punctures (approx. $1\frac{1}{2} \times$); B, an egg (approx. $20 \times$); C, a section through an egg puncture showing egg in place and the plug of feces with which the hole is closed (approx. $16 \times$). (After Parrot and Fulton.)

127). A drop of mucilaginous fluid is secreted on top of the egg. *O. niveus* covers the egg and plugs the hole with a pellet of feces, and *O. angustipennis* uses a pellet of chewed bark, usually taken from diseased tissue (Fig. 128).

Tree crickets are omnivorous feeders, eating any organic material of sufficient softness, including soft insects such as aphids and scale insects. They also feed on decaying plant tissue and the associated fungus spores and mycelium. Microscopic examination of adult tree crickets shows abundant spores of many fungi on all parts of the body. Viable spores of many species of fungus, including *L. coniothyrium*, are found abundantly in the intestinal tracts and in fecal pellets. From these facts, it is evident that the

act of oviposition is an effective means of inoculating the apple twigs with the pathogenic fungus. Not all oviposition punctures result in cankers, but enough of them are infected to make the insect a vector of major importance. The eggs do not hatch until June of the year following oviposition, and there is only one brood in the region of New York.

Because of the omnivorous feeding habits of the insects and their preference for soft decayed tissues, it is probable that they are to some extent vectors of other diseases. Garman in Kentucky, as early as 1904, reported brown rot of peaches developing from the feeding wounds of tree crickets on peaches and plums and black rot from feeding wounds on grapes. Urbahns (1923), also, reported destructive outbreaks of brown rot in California following the feeding punctures of these insects. Both the feeding and breeding habits of the tree crickets make them potential vectors for many plant diseases. They should be looked upon with suspicion when they are found associated with almost any plant disease.

Chestnut Blight.—The *Endothia* canker of chestnut, or chestnut blight, was first discovered in one of the parks of New York, in 1904, presumably having been introduced from the Orient on imported Japanese chestnut trees. It is caused by *Endothia parasitica* (Murr.) Anderson and Anderson, an Ascomycete of the order Sphaeriales. The fungus produces a rapidly developing canker of the twigs, branches, or trunk of the tree. Infection is usually fatal, leading to the death of the tree in a relatively short period of time. The chestnut-blight pathogen has been thoroughly described by Shear, Stevens, and Tiller (1917). The disease has spread rapidly throughout the range of the chestnut in the eastern United States; in the course of less than 25 years after its discovery; it had destroyed almost the entire stand of native chestnuts in North America. A strenuous campaign was waged to eradicate and check the spread of the disease, but all efforts were in vain. It spread so rapidly that no practical means of checking it was ever discovered. In the course of this eradication campaign, it is only natural that considerable attention should have been given to the various methods of spore dissemination which enabled the disease to spread so quickly.

From the very beginning of the study of the disease, insects were considered as important agents in disseminating the spores,

but the idea was based largely on observations and inference. Rankin (1912) stated that "reasoning by analogy with what is known of the behavior of many fungi, such agencies as borers, birds, ants, and the wind, etc., have been suggested but in no wise proved to be responsible." The most extensive study of insects as possible vectors of the disease was made by Studhalter and Ruggles (1915). Heald, Gardner, and Studhalter (1915) demonstrated that although the ascospores were readily dis-



FIG. 129.—A branch affected with chestnut canker showing the extrusion of spores from the pycnidia in long sticky "spore horns." These spores adhere readily to insects and birds and may be carried for long distances by them. Local dissemination is accomplished most readily through rain water. (After Heald.)

seminated by wind, the pycnidiospores were not so well adapted to wind dissemination, being extruded in long, sticky spore horns that become hard on drying (Fig. 129). The spores were shown to be better adapted to dissemination by water and insects. Studhalter and Ruggles studied the spores found on about seventy-five individual insects representing about twenty-five different species, all taken from chestnut-blight cankers. They found that about 30 per cent of them carried spores on their bodies, and they concluded that the insects were of importance as vectors of the disease.

The conclusions of Studhalter and Ruggles, however, were strongly criticized by Craighead (1916), who pointed out that most of the insects on which spores were found rarely visited or attacked living trees and for this reason could be of little significance as vectors of chestnut blight. He maintained that other insects not studied by Studhalter and Ruggles would be more likely to transmit the disease. The most likely insect vector according to Craighead is the long-horned beetle (*Leptura nitens* Forster) which bores in the bark of over 90 per cent of the living chestnuts over 10 inches in diameter. He states that this species has adapted itself to breeding in great numbers in chestnut-bark cankers, a fact which suggests strongly that it is an important vector. It is to be regretted that this insect was not studied more thoroughly.

Craighead claimed that the beetle *Leptostylus macula* Say, considered by Studhalter and Ruggles as the most important vector, rarely ever visited living trees and could not be of significance in transmitting the disease to healthy trees. On the other hand, Craighead (1912) believes that this insect, by eating the spores and preventing their dissemination, retards rather than aids the disease in its spread. *Leptostylus macula* may be of no significance as a vector, but it is difficult to believe that the quantity of spores eaten by the insect would be a significant check to the spread of the disease. Craighead recognized the possible importance of many species of insects in making fresh wounds through which spores disseminated by wind could infect. He states that such wounds have been found infected and expresses the belief that the making of wounds, through which wind-blown spores infect, is the chief role of insects in the epiphytology of chestnut blight.

Chestnut blight spread so quickly over the entire range of the chestnut, as stated above, that all efforts to control it were discontinued. Thus, interest in the study of the method of its spread also waned, and the problem of insect transmission of the disease was left only partly solved.

Heald and Studhalter (1914) demonstrated that several species of bird were also vectors of the chestnut-blight pathogen, suggesting that migratory species could be responsible for long-distance dissemination and new centers of infection. The fungus is well adapted to rapid dissemination by birds and many other agencies,

but the relative importance of the different agencies of dissemination has never been determined. For a more detailed discussion of the role of birds as vectors of chestnut blight, see Chap. XI.

Insects and Tomato Leaf-spot Diseases.—Martin (1918) has investigated the role of several leaf-eating insects in the dissemination of spores of *Septoria lycopersici* Speg. and *Alternaria solani* (E. and M.) Jones and Grouet, causing leaf spots of tomato. It was first observed that flea beetle punctures frequently formed the center of an early blight infection. Flea beetles (*Epitrix cucumeris* Harris) collected at random in tomato fields were found to carry viable spores both externally and in their intestinal tracts. It was demonstrated that the spores of both fungi pass through the alimentary canal and survive uninjured in fecal deposits. Similar relationships were demonstrated for the Colorado potato beetle (*Leptinotarsa decimlineata* Say) and the tomato worm (*Protoparce carolina* L.).

Unfortunately, neither disease was experimentally transmitted by the insects concerned. The conclusion that they serve as vectors is based on circumstantial evidence only.

It was further shown that pickers carry great quantities of spores on their hands and garments, and Martin concludes that much of the inoculum is disseminated in this way. Pickers are considered to be of more importance than the insects, which are of greatest importance in the dissemination of inoculum early in the season.

Insects and Sooty Mold.—Many crop plants, especially those of tropical or subtropical regions, are often severely affected with a disease known as "sooty mold," caused by a number of different species of fungi possessing dark-colored mycelium. One of the most common of the sooty mold fungi is *Capnodium citri* which causes the troublesome sooty mold of citrus fruits. The fungi grow as black velvety membranes over the surfaces of leaves and fruit (Fig. 130). They are not true parasites, for they do not penetrate the cells of the plant but live entirely on the surface and derive their nourishment from honeydew secreted by insects. Even though the sooty mold does not parasitize the plant, it severely injures the tree by forming a mat of black mycelium that covers a large proportion of the leaf surface and thus interferes with the processes of photosynthesis and respiration. This results in lower yields of poor-quality

fruit and makes the trees less resistant to drought or other adverse factors. The fungus also may disfigure the fruit and appreciably reduce its market value. Any insect that secretes honeydew in sufficient quantity may meet the requirement of the sooty-mold fungus. Scale insects, aphids, and the larvae of white flies are most frequently associated with sooty molds.

One of the best descriptions of the association has been given by Webber (1897), who described in detail the sooty mold of orange in Florida. Here the fungus was associated with the white fly (*Aleyrodes citri* R. and H.), the wax scale (*Ceroplastes floridensis* Comstock), several species of *Lecanium*, the mealy bug (*Dacty-*



FIG. 130.—A citrus leaf affected with sooty mold. Some of the mold has been stripped off exposing the leaf surface. (After Rhoads.)

lodus citri Risso), and the aphid (*Aphis gossypii* Glov.). Of these insects, the white fly (*A. citri*) was by far the most important.

The insects for the most part feed upon the lower surfaces of the leaves. The honeydew secreted by them falls principally on the upper surface of the leaves and fruit immediately underneath, and it is here that the sooty mold grows most luxuriantly. Several different kinds of spore are formed by the sooty-mold fungi, most of which are readily disseminated by wind. There is no evidence that they are disseminated more than incidentally by the insects furnishing the honeydew on which they grow.

Because the black mold is dependent upon the insects for its nourishment, the most effective means of control has been indirectly through the control of the insects. This has been

accomplished by spraying with insecticides, by fumigation with hydrocyanic acid gas, and by the use of natural enemies such as entomogenous fungi and insect parasites. Practical experience has shown that when the insects are destroyed the sooty-mold fungi disappear, thus confirming the dependence of the fungi upon the insects for nourishment.

Insects and Brown Rot of Stone Fruits.—Brown rot caused by *Sclerotinia spp.* is one of the most destructive diseases of stone fruits, causing enormous losses by decay of the fruit as well as through blossom and twig blight. The disease is almost world-wide in its distribution, and there are few places where peaches can be grown successfully without spraying to control the rot.

The spores of the brown-rot fungus are commonly wind-borne; and although the pathogen may penetrate the skin of uninjured fruits through stomata and lenticels, infection is facilitated by wounds. The feeding and oviposition wounds made in the fruit by the plum curculio (*Conotrachelus nenuphar* Herbst) are very frequently portals of entry for the fungus (Scott and Quaintance 1911). It is generally assumed that wind-blown spores are responsible for the infection that takes place through the insect punctures, but as the curculios have abundant opportunity of becoming surface-contaminated with spores it is very probable that they also disseminate the spores and introduce some of them into the freshly made wounds.

The significance of the curculio as a vector of brown rot is more evident in orchards sprayed with lime sulphur where infection by wind-blown spores is reduced to a minimum. In one experiment reported by Scott and Ayres (1910), 93 per cent of the fruit infection could be traced to curculio wounds.

In view of the importance of the curculio in brown-rot infection, it is obvious that a sound control program for brown rot must include provisions for the control of the curculio.

The Potato Flea Beetle and Potato Scab.—Scab caused by *Actinomyces scabies* (Thaxter) Güssow is one of the most prevalent diseases of the potato. The pathogen lives indefinitely in the soil and infects the developing tubers, causing the well-known corky scab pustules. Ingression is chiefly through the lenticels; but wounds also may serve as infection courts.

MacMillan and Schaal (1929) have demonstrated that the scab pathogen commonly infects the tissues injured by the larvae

of the potato flea beetle (*Epitrix cucumeris* Harris). In the absence of scab, the injury caused by the flea-beetle larvae consists of inconspicuous "worm tracks" with a smooth surface, differing only in color from the normal periderm. When infected by scab, the worm tracks are raised, bulging, and eruptive, seriously decreasing the market value of the tubers. In other cases, the lesions caused by the flea beetles are invaded by *Rhizoctonia solani*, a fungus that usually does not infect the tissues of the tuber.

Schaal (1934) investigated further the association of scab with flea-beetle injury and has demonstrated that flea-beetle larvae, collected from potato soil, carry *Actinomyces* both externally and internally. The pathogen was not found in or on the eggs of the beetles when deposited in sterilized cages. The insect in all probability plays no part in the dissemination of the pathogen but is an effective agent of inoculation, a large part of the injury being caused by the scab pathogen that it introduces.

Blackleg of Cabbage and the Cabbage Maggot.—Blackleg of cabbage and other crucifers is caused by a fungus [*Phoma lingam* (Tode) Desmaz.]. It causes a dry, black necrosis of the roots and stem, often killing the plant in relatively early stages of development. It also may cause a spotting of the leaves and a decay of the cabbage head in storage. The disease was first studied extensively by Ritzema Bos (1906) and Quanjer (1907) in Holland. Quanjer concluded that the fungus could not infect the roots unless they were injured and that the injuries made by the cabbage maggot (*Hylemyia brassicae* Bouche) were the principal avenues of entrance. His belief that the fungus was not seed-transmitted caused him to place special emphasis on maggot infestation as a prerequisite for infection. Henderson (1918), however, has shown that the fungus is readily seed-transmitted and the roots and stems may be infected without previous injury. These results naturally raise the question of the importance of maggot infestation in the epiphytology of the disease. Unfortunately, Henderson did not investigate this aspect of the disease extensively. Even though the maggot injury may not be necessary, Quanjer's observations indicate that it may be a significant contributory factor. There is need for a more careful study of the relationship of insect injury to

blackleg infection and development in light of the more recent information about the parasitism of the fungus.

Insects and the Red Rot of Sugar Cane.—Red rot, caused by *Colletotrichum falcatum* Went., is one of the most destructive diseases of sugar cane in the United States. It causes severe rotting of the stalks, stubble rhizomes, and leaf mid-ribs. It has been the cause of failure of a number of the leading commercial varieties of sugar cane. A complete account of the disease in Louisiana and Florida has been published by Abbott (1938).

Red rot is transmitted in several different ways. Spores are produced abundantly and are widely disseminated by the wind. Although infection may occur by direct penetration of the epidermis, the most destructive phases of the disease are those following infection through wounds. According to Abbott, infection of the stalk, in Louisiana and Florida, occurs principally through the tunnels of the moth borer (*Diatraea saccharalis* F.). Injuries made by the sugar-cane weevil (*Ana-centrus* sp.) also may serve as points of infection for underground parts of the stalk. Abbott concludes that infection of the leaves occurs most frequently through insect wounds, although direct penetration of uninjured epidermis is recognized. No studies of the detailed relationships between the insects and the fungus have been reported. One would assume from much of the published statements that infection is accomplished by wind-blown spores which enter the wounds made by the insects, but in all probability careful study would show that the spores are introduced by the insects in their feeding activities.

Insects and Diseases of Mushrooms.—Cultivated mushrooms are subject to a number of destructive diseases, several of which are transmitted extensively by insects. The most important of these are "bubbles" and "plaster mold." The former is caused by a fungus (*Mycogone* sp.) that greatly distorts the mushroom, causing it eventually to become soft and decayed. Plaster mold is caused by a fungus that does not parasitize the mushroom directly but grows throughout the compost and prevents the normal development of the fruiting bodies. According to Charles and Popenoe (1930), spores of both these fungi are disseminated by the various species of fungus fly that infest the mushroom. Charles states:

In houses where these diseases occur mushroom flies have been examined which carried on their somewhat spiny bodies and legs hundreds of spores of these fungi. . . . These flies move actively about through mushroom houses, both by walking and by flight, and are undoubtedly responsible for the rapid increase in the distribution of the diseases in large houses. Diseased mushrooms develop an odor likely to attract some of the larger carrion flies from the outside and these larger flies are capable of carrying much greater numbers of spores throughout the house.

The control of both diseases is aided by adequate control of the insect vector.

Plum Wilt and the Peach-tree Borer.—Plum wilt, a destructive disease of plum trees in Georgia, was described in 1916 by Higgins, who concluded that wounds made by the peach-tree borer (*Aegeria exitosa* Say) were the most important avenues of infection. Plum wilt is caused by the fungus *Lasiodiplodia triflorae* Higgins, which is a vascular parasite affecting principally the conducting elements and the medullary rays in the wood. The wilting results from the inhibition of the water movement by gum deposits.

The fungus cannot penetrate the uninjured bark and is entirely dependent upon wounds. Although mechanical injuries and wounds made by other agencies may serve as portals of entry, the wounds made by the borer appear to be of greatest importance.

The disease is more destructive on the Japanese plums and hybrid varieties of Japanese-plum parentage. One of the reasons for this varietal preference was attributed by Higgins to the use of peach stock on which to bud the plums. The peach stock is more attractive to the borer than plum wood, and infestation that takes place in the peach stock spreads readily into the plum wood.

The fungus fruits on affected trees near the surface of the soil where the bark is continually moist, and the spores are extruded from the pycnidia in white masses or in long gelatinous tendrils. The method of dissemination of the spores is not fully known, and the question of whether or not the spores are disseminated by the adult borer has been left unanswered.

The peach-tree borers are Lepidopterous insects. They hibernate in the soil or in the bark of infested tree trunks in the pupal stages, emerging as adult moths in early spring. Eggs

are deposited on the surface of the bark of Drupaceous trees near the base of the trunk. The young larvae bore through the bark soon after hatching and complete their development in the inner bark of the roots or the base of the trunk. Although many varieties of Drupaceous tree are attacked, the borer is primarily a pest of the peach or of trees grafted on peach stock.

Insect Dissemination of the Cotton-wilt Pathogen.—*Fusarium* wilt caused by *Fusarium vasinfectum* Atk. is a destructive disease of cotton throughout the southern part of the United States. In common with other pathogens of this kind, it spreads relatively slowly through the soil. The appearance of the disease in new localities in small areas in the field is not uncommon, indicating that aerial dissemination of inoculum occurs in some way. It is not always possible to determine the origin of these local infestations, and the method of aerial dissemination has been a subject of much speculation. Taubenhaus and Christensen (1936) have investigated the survival of *F. vasinfectum* in the intestinal tract and fecal pellets of numerous insects that normally feed on cotton plants. The fungus was recovered in viable condition from the fecal pellets of nearly all the species tested, except those which feed normally on decaying organic matter (*Collembola*, etc.). The following grasshoppers were found to act as agents of dissemination of the fungus: *Melanoplus differentialis* Thos., *M. mexicanus* Sauss., *M. femur-rubrum* De G., *Encoptolophus texensis* Brun., *Spharagemon cristatum* Scudd., *Tomonatus aztecus* Sauss., *Chortophaga viridifasciata* var. *austrator* De G., *Schistocerca americana* Drury, *S. obscura* Fabr., *Trimentropis citrina* Scudd., and *Dissosteira carolina* L. These insects were considered of importance in field-to-field spread of the disease. All evidence indicated that the association between insect and fungus is entirely mechanical and that the passage of the fungus through the alimentary tract in the form of spores and mycelial fragments is relatively rapid.

Monochamus spp. and Heart Rot of Conifers.—The relationship of wood-boring beetles and wood decay in logs of red pine has been studied by Leach, Orr, and Christensen (1937). When freshly cut logs were exposed to insect infestation, bark beetles were the first insects to attack them. The bark beetles were soon followed by various other insects, including several wood-boring species. As soon as the logs became infested with insects,

the sapwood was rapidly decayed by fungi. The most prevalent fungus was *Peniophora gigantea* (Fr.) Masee, which eventually spread to the heartwood, causing a reddish brown decay. It was demonstrated that there was a definite correlation between the amount and rapidity of heartwood decay and the infestation by *Monochamus scutellatus* (Say) and *M. notatus* (Drury). All

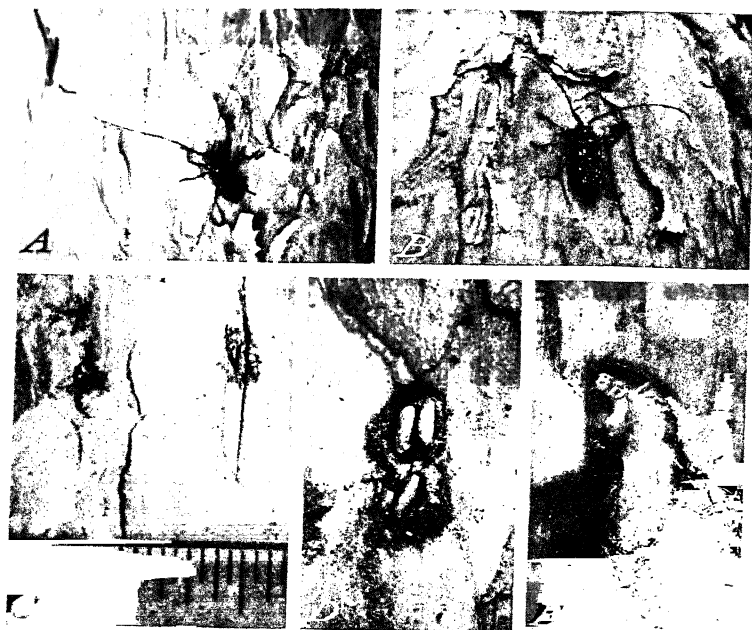


FIG. 131.—*Monochamus scutellatus*, a wood-boring insect that aids a wood-rotting fungus in the invasion of the heart rot of conifers. *a*, a male; *b*, a female cutting an egg niche through the bark; *C*, egg niches; *D*, eggs in the inner bark; *E*, a larva feeding on the inner bark and outer sapwood.

evidence indicated that the beetles were not responsible for introducing the fungus into the log but that they aided the fungus in the invasion of the heartwood and so hastened its destruction.

The beetles (Fig. 131) lay their eggs in the inner bark through niches made with their chewing mouth parts. On hatching, the larvae feed for some time on the inner bark and outer layers of sapwood where they become contaminated with the wood-rotting

fungus. As the larvae approach maturity, they bore deep into the heartwood, later returning to the sapwood to pupate (Fig.

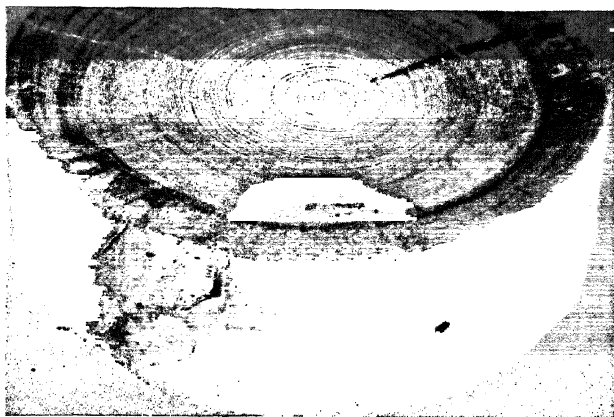


FIG. 132.—A section through a red-pine log showing the decay associated with the larval tunnel of *Monochamus scutellatus* in the heartwood. The larva entered the wood through the ovoid hole at the left, and the mature beetle emerged from the round hole at the right.

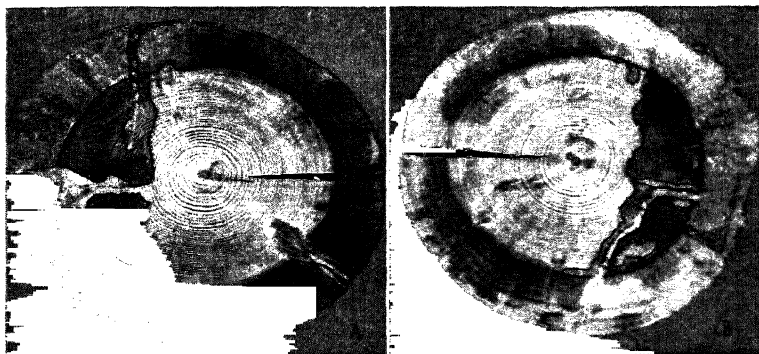


FIG. 133.—Two sections of the same experimental red-pine log showing two areas of decay, each arising from a larval tunnel of *Monochamus scutellatus*.

132). Observations on experimental logs showed that the greater part of the heartwood decay always spread from the larval tunnels (Figs. 132 and 133).

The fungus spreads through the wood in a longitudinal direction much more rapidly than in a radial or tangential direction. The larvae take the fungus deep into the wood radially and tangentially after which it spreads rapidly up and down the log.

Several species of buprestid beetle also attack the logs, but they apparently do not influence greatly the development of heart rot. The larvae keep their tunnels packed with frass and do not move about in them. The *Monochamus* larvae, however, keep their tunnel open and move back and forth frequently in disposing

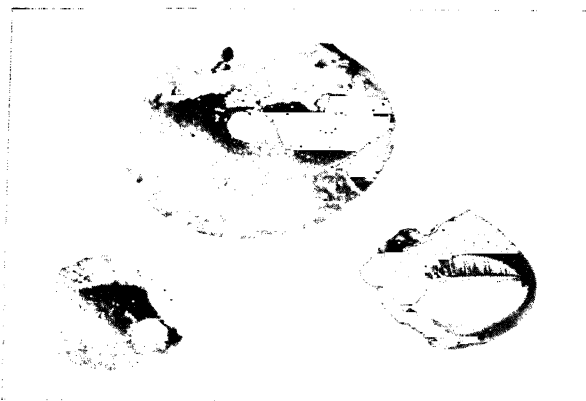


FIG. 134.—Three fruiting bodies of *Polyporus volvatus*, showing the small opening through the membrane that encloses the hymenial layer forming a chamber in which the spores accumulate. The sporophore on the right has been cut in section in a vertical plane through the opening and shows the internal structure.

of their frass. This difference in habits may explain in part the difference in their influence on the rate of decay.

Insects and a Sapwood Decay of Conifers.—The sapwood of trees of various coniferous species is sometimes decayed by *Polyporus volvatus* Pk. The decay affects dead or dying trees, especially those infested with bark beetles. The fruiting bodies nearly always grow out of the holes made through the bark by the beetles, and there is considerable evidence to show that the spores are introduced into the sapwood by the beetles (Hubbard 1892). The fruiting surface of the sporophore is at first completely covered with a leathery membrane continuous with the upper surface. As the sporophore matures, a small opening is formed in

the center of the membrane. The spores are shed within the cavity and are not subject to wind dispersal (Fig. 134). However, beetles and other insects enter the hole and, in crawling about within the enclosed space, become contaminated with the spores which they presumably introduce under the bark as they infest new trees. Hubbard has observed more than a dozen different species of insect in the spore chamber or feeding upon the sporophores. The bark beetles, because of their habit of boring into the bark, have the greatest opportunity of inoculating the sapwood and are generally considered to be the most important vectors.

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CHAPTER VIII

INSECTS AND VIRUS DISEASES

The viruses that affect plants cause some of the most destructive diseases of agricultural crops. Early records reveal that virus diseases were prevalent on certain cultivated crops more than three hundred years ago; yet the full extent of the damage caused by them was not recognized until after the close of the last century. There is indeed much evidence for the view that the virus diseases as a group have become more prevalent and destructive in recent years. This may be explained to some extent by the more general recognition of virus diseases following the extensive study of the past 25 or 30 years. On the other hand, the great increase in commerce and travel and the exchange of plant materials would favor the spread of viruses to new regions. Moreover, it is a recognized fact that commerce has resulted in distributing many insect vectors to new regions throughout the world.

It must be recognized as a possibility also that viruses which originally affected only wild plants may have become more destructive to cultivated plants. An increasing amount of the land has been brought under cultivation with a corresponding destruction of the weed hosts of both the viruses and their insect vectors. It is only reasonable to suppose that as this change occurred the insect vectors and the viruses transmitted by them would become more prevalent on the cultivated crops. This is especially true of such viruses as curly top of sugar beet, aster yellows, and spotted wilt of tomato, each of which has a very wide host range among both weeds and cultivated plants.

A relatively large number of virus diseases of plants have been described in the past 25 years, and new ones are being reported at very frequent intervals. Extensive bibliographies on virus diseases have been published by Atanasoff (1934, 1937), Otero and Cook (1934, 1935, 1936), Cook (1935*a*, 1936*a*), and Smith (1937*a*). Cook (1935*b*, 1936*b*) has published, also, an extensive

index of insect vectors of virus diseases. Almost all cultivated crops are affected by one or more viruses. At least 25 different viruses have been reported as affecting the potato (Folsom and Bonde 1936), and more than a dozen are known to attack tobacco. It is probable that the same virus has been described under different names on different host plants. A single plant also may be affected with more than one virus, resulting in a confusion of symptoms that makes identification difficult. Many of the virus diseases, originally thought to be caused by a single virus, on further study have been found to be caused by two or more viruses that could be separated by appropriate methods. The number of viruses recognized as distinct are so numerous that no effort will be made here to discuss them all, not even all those known to be transmitted by insects. A relatively complete descriptive list of the known plant virus diseases has been published by Smith (1937a).

The identification of the various viruses is difficult because their true nature is still uncertain. For many years, plant pathologists have been inclined to accept the hypothesis that viruses are ultramicroscopic microorganisms living as obligate parasites. However, the more recent works of Vinson (1936), Vinson and Petre (1929, 1931), Stanley (1936a, b, c, 1937b, 1938a, 1938b), and others furnish evidence that the viruses may be inanimate and consist of proteins of high molecular weight which, in the presence of living cells, stimulate the production of more protein of the same kind. There are some objections to the concept of viruses as inanimate proteins, as pointed out by Rawlins and Takahashi (1938), Bawden and Pirie (1937), Gortner (1938), and others; yet the evidence is strongly in favor of the protein hypothesis. A final solution of this problem will greatly facilitate the study of virus diseases as well as their relationships with insect vectors.

AGENTS OF VIRUS DISSEMINATION

No group of plant pathogens is more closely associated with insects than the viruses. There are very few, if any, true virus diseases that are not to some extent transmitted by insects, and there are many that are transmitted in nature only by insects. Despite the importance of insects as vectors of virus diseases, there are several other ways in which viruses may be spread. In

many cases, a single virus may be transmitted by several different agencies, and the evaluation of the relative importance of each agent is an important factor in the control of virus diseases. In addition to being transmitted by insects, virus diseases may be spread in nature by wind, water, seed, and vegetative propagation. They may be transmitted artificially by inoculation and by grafting.

Wind.—Very important in disseminating fungus spores, wind is of relatively little significance in the spread of virus diseases. Some viruses, however, such as that of tobacco mosaic are very resistant to desiccation and aging and may be disseminated by wind in the form of dried and crumbled leaf tissue. Because the tobacco-mosaic virus infects so readily through the slightest injury, it is very likely that wind is of some importance as a vector; but, in general, dissemination in this way is relatively unimportant when compared with insect dissemination.

A new virus disease of tobacco, tobacco necrosis (*Nicotiana virus 11*),¹ has been described by Smith (1937 *c, d*), who presented evidence to show that the virus is air borne. Broad beans, which are also very susceptible to the virus, were infected when inoculated with previously sterile water through which air from the greenhouse containing infected tobacco plants was drawn. Caldwell (1937) has objected to some of the conclusions reached by Smith, pointing out that the origin of the air-borne virus particles was not known and that infection by particles directly from the air had not been proved beyond question. Price (1938) in an extensive study of this virus was unable to obtain any evidence that it was air borne. No natural transmission was observed in his greenhouse experiments although old infected plants were present in the same house. Smith, on the other hand, reported frequent and rapid spread which he interpreted as caused by air-borne particles of virus. He concludes that the virus particles were washed into the soil, from which they gained entrance into the roots.

Matsumoto (1938) reported another disease of tobacco and presented data that led him to believe that a virus was transmitted through the air directly from a diseased to a healthy plant. In a footnote in a later paper, however, Matsumoto (1938) throws some doubt on the correctness of his earlier con-

¹ Classification of Kenneth M. Smith.

clusion by reporting the presence of some very small tyroglyphid mites on his experimental plants. It appears probable that he was not working with a true virus disease. The importance and significance of the problem of wind transmission justifies more extensive study under carefully controlled conditions; for if Smith's conclusions are proved correct, a new means of virus transmission will have been established. Extreme caution must be used, however, in drawing final conclusions. Every other possible means of transmission must be eliminated by rigidly controlled experiments. It would appear that a highly infectious virus could be readily transmitted by contact in handling the plants. These factors must be carefully controlled in experiments of this kind.

Wind may be concerned also in so far as it may be a factor in the dissemination of the insect vectors. As pointed out in Chap. XV, there is evidence that certain insects may be blown by wind for long distances. In general, however, it may be concluded that wind is a minor factor in the dissemination of viruses.

Water.—Water does not seem to be an important agent of virus dissemination. In violent wind and hail storms in which there is much wounding of tissue, wind and water combined theoretically could be responsible for much local spread of certain virus diseases. Moreover, when infection takes place underground, the soil water probably plays an important part in transmitting the virus. The significance of this type of spread is very difficult to evaluate, because it has not been the subject of much experimentation. Tobacco necrosis (*Nicotiana virus 11*), according to Smith (1937*b*, *c*, *d*), is readily transmitted by water to the roots, through which infection occurs without the aid of artificial wounding. This virus appears to be highly infectious and may be transmitted by contact, although it is difficult to transmit by grafting on most host plants.

Guttation water apparently is of little significance in the transmission of viruses because, as shown by Caldwell (1930, 1931), viruses are rarely present in guttation drops even though the plant may be heavily infected. The virus apparently is unable to diffuse through the protoplasmic membrane into the tracheae. Furthermore, when viruses are placed in the guttation drops, infection does not occur unless the adjacent plant cells are wounded.

Soil.—The term “soil transmission” is often used to denote a specific method of virus transmission, but there appears to be little or no justification for using the term in this sense. The soil is not an agent of dissemination, and the term merely covers a great amount of ignorance about the actual means of spread. Infection may take place through underground parts of a plant and be transmitted by insects working underground, by root contact, by soil water, or by direct inoculation through cultivation injury. Because of the difficulty of observing the underground plant parts, the actual mechanism of underground infection is usually obscure; but there is no reason for supposing that it is fundamentally different from infection through the aboveground parts of the plant.

There is very little evidence to indicate that underground infection is common for many of the virus diseases. Webb (1927, 1928), however, has shown that in wheat mosaic infection may occur underground through either the roots or the crown, or both. Infection through parts of the plant aboveground apparently does not occur in nature. Wheat mosaic is transmissible by artificial sap inoculation, but no insect vector is known. The virus survives in the soil for an indefinite time and is not removed by thoroughly and repeatedly washing the soil. Neither the exact point of entrance through the roots or the crown nor the method by which entrance is accomplished is known. There is need for more thorough investigation of the ability of viruses to survive in the soil and the frequency of infection through roots and other underground parts of plants.

Root infection of tobacco from contaminated soil has been reported by Lehman (1934) and by Johnson (1937), but the mechanism of infection was not determined. Smith (1937*a, b*) also has shown that tobacco plants may be infected through the roots by the virus of tobacco necrosis from contaminated soil water. However, the virus was not able to infect the roots of plants grown in water cultures contaminated with the virus. This fact would suggest the possibility that injuries caused by soil particles furnished the necessary wounds for infection. Evidence leading to the same conclusion has been presented also by Price (1938).

Seed.—Seed transmission of viruses is known to occur, but it is not the rule. Perhaps the best-known and most striking case is

that of bean mosaic. Seed transmission of the bean-mosaic virus to the extent of 50 per cent has been obtained under experimental conditions. Seed transmission has been studied extensively by Nelson (1932) and Nelson and Down (1933), who have shown that seed infection of beans is very erratic, only a part of the seed on a badly diseased plant becoming infected. They have shown that often only a few seeds in a pod may transmit the virus and that the position in the pod is not the determining factor.

Viruses appear to be more readily transmitted through the seeds of legumes than those of many other plants, but seed transmission is by no means confined to this family. Other examples have been reported by Doolittle and Gilbert (1919) in seed of wild cucumber, Kendrick (1934) in muskmelon seed, Elze (1931a) in potato seed, Newhall (1923) in lettuce seed, Berkeley and Madden (1932) and Doolittle and Beecher (1937) in tomato seed, and Henderson (1931) in petunia seed. For other examples of seed transmission of viruses, see the bibliography on seed-borne parasites by Orton (1931).

The factors governing seed infection are not very well known. It is difficult to understand why it occurs in some plants and not in others and why only part of the seeds on heavily infected plants carry the virus. These occurrences would seem to indicate that the viruses are not so universally distributed in the tissues of the plants as has been generally believed.

Pollen.—Where seed transmission occurs, it has been assumed generally that the seeds become infected systemically from the infected mother plant. Another means of seed infection has been pointed out by Blakeslee (1921) who demonstrated that a virus on *Datura stramonium* is transmitted to the seed by pollen from infected plants in as high as 79 per cent of the seed. Reddick (1931) and Nelson and Down (1933) have shown that infected bean seed may be accounted for in this way. The possibilities of this method of transmission have not been extensively investigated. There is need for further work.

Other Plant Parts.—Those plants which are vegetatively propagated provide one of the most effective means of virus dissemination and perpetuation. Recovery is rare in virus diseases of plants, and vegetative propagation from infected plants, whether by tubers, corms, cuttings, grafts, or any other method, usually results in 100 per cent infection of the progeny. Were it

not for the fact that many virus-infected plants yield progressively less until they fail to reproduce themselves and are eliminated, the percentage of infection in vegetatively propagated crops would eventually approach 100 per cent. This apparently is what has happened in the case of the latent virus of potatoes. Those varieties of potatoes in which the virus produces no ill effect, and consequently no tendency toward elimination of infected plants, have, in time, become universally infected.

Parasitic Fungi.—The possibility of virus transmission through the agency of parasitic fungi or bacteria has been suggested (Nelson 1932). Theoretically it would be possible for spores of a parasitic fungus, produced on a virus-infected plant, to harbor the virus and introduce it into the cells of a healthy plant following germination and infection. This possibility appears remote, and no positive experimental evidence is available. Nelson (1932) observed that bean rust [*Uromyces appendiculatus* (Pers.) Rev.] taken from beans infected with mosaic did not transmit the virus when transferred to healthy plants.

Man.—Man himself is one of the most effective agents of virus dissemination. In the sale and distribution of seeds and plant parts and in pruning and other cultivating operations, viruses are disseminated widely. Valleau and Johnson (1927) have shown that tobacco mosaic may be spread by workers who chew or smoke tobacco while working with the tobacco plants. Many viruses are readily transmitted artificially by direct sap inoculation, but some are very difficult to transmit in this way. Still others have been artificially transmitted only by grafting. The majority of viruses are readily transmitted by grafting even though they may not be transmitted by other means.

In a consideration of the transmission of virus diseases, there are a few fundamental facts that should be kept in mind. First, it should be remembered that there is no multiplication of the virus in the absence of living cells, in either plant or insect. Consequently it appears that the virus inoculum must come in contact with the protoplasm of the plant cell before infection occurs. This fact has led to the tacit assumption that some sort of wound is always necessary for virus infection. Sheffield (1936a), working with tobacco, tomato, *Nicotiana glutinosa*, and *Solanum nodiflorum*, presented evidence to show that tobacco-mosaic virus 1 and tobacco virus 6 cannot enter the

plant unless some of the surface cells are injured. Duggar and Johnson (1933), however, secured infection of tobacco plants by spraying the leaves with a suspension of tobacco mosaic virus. Since no wounds were made or could be detected, stomatal infection appears to be the only explanation. Drake *et al.* (1934) and Sheffield (1936*b*) have emphasized the importance of plasmodesmata in infection and invasion of plant tissues by viruses. If virus particles entering through the stomata come in contact with plasmodesmata of the loosely arranged substomatal parenchyma, stomatal infection is more easily explained. Finally, one should bear in mind that there is much difference in the properties of different viruses, especially in regard to the influence of the extracellular environment. In all probability, there is as much variation among the plant viruses as there is among the plant pathogenic bacteria.

Insects.—Practically all the insect vectors of virus diseases are found in the following five orders.

1. Orthoptera (grasshoppers).
2. Thysanoptera (thrips).
3. Homoptera (aphids, leaf hoppers, white flies, and scale insects).
4. Hemiptera (lace bugs and other plant bugs).
5. Coleoptera (beetles).

1. *Orthoptera*.—The insects of this order all have chewing mouth parts, and they are as a rule not very effective as vectors of virus diseases. Spindle tuber of potatoes, a virus disease that is transmitted very easily by artificial inoculation, has been shown by Goss (1928, 1929, 1931) to be transmitted in nature by several species of grasshopper (*Melanoplus spp.*). The transmission apparently is entirely mechanical. Not many insects of this order have been incriminated as vectors of virus diseases.

2. *Thysanoptera*.—This order includes the thrips. These are small insects with asymmetrical mouth parts, and they feed with a rasping-sucking action. Thrips (*Frankliniella lycopersici* Andr. and *Thrips tabaci* Lind.) have been proved to be vectors of the spotted wilt of tomato, pineapple yellow spot, and a streak of peas (Samuel, Bald, and Pittman 1930; Linford 1931*a, b*, 1932). These diseases eventually may be shown to be identical. Transmission here is definitely biological and probably specific. Although the ability of thrips to transmit other viruses has not been thoroughly investigated, it is probable that they may be

involved in the transmission of other virus diseases, where the relationship is less specific and the method of transmission purely mechanical. However, because of the very shallow feeding wounds made by thrips, they would be of little importance as vectors of viruses that are localized in the phloem.

3. *Homoptera*.—This is by far the most important order of insects in regard to the transmission of virus diseases. Approximately 90 per cent of all the insects concerned in the transmission of plant viruses are members of this order. The vectors are found chiefly in the following families: Aphididae (aphids, or plant lice), Cicadellidae (leaf hoppers), Fulgoridae (plant hoppers, or lantern flies), Aleyrodidae (white flies); and a few doubtful cases have been reported in the Coccidae (scale insects and mealy bugs).

The Homoptera all have piercing-sucking mouth parts and feed exclusively on plant sap. Their method of feeding makes them especially effective as vectors of virus diseases. Their long narrow beaks inject the inoculum deep into the tissues without wholesale destruction of cells. Since the virus can multiply only in the presence of living cells, this is a decided advantage. Insects that kill large numbers of cells around the feeding wounds would be less effective in bringing the virus in contact with living protoplasm. Many insects of this order, which cause very little obvious direct injury to their host plants, are now considered pests of major economic importance because of their role in transmitting virus diseases. A demand for more effective control of these species has followed the discovery of their role as vectors.

The *aphids*, or plant lice, include a large number of vectors and transmit more different virus diseases than do the members of any other insect family. More than twenty different species of aphids have been shown to be concerned in the transmission of a considerably larger number of different viruses. Certain species such as *Myzus persicae* (Sulz.) and *Macrosiphum gei* (Kalt.) are known vectors for a dozen or more different viruses, while other species are more limited in this respect. In general, there appears to be somewhat less specificity between the aphids and the viruses they transmit than there is between the leaf hoppers and the viruses spread by them. Nevertheless there is abundant evidence of certain types of specificity among the

aphid vectors. On the other hand, there are numerous examples of a single species of aphid transmitting several different viruses, while such cases are rare among the leaf hoppers.

The leaf hoppers rank next to the aphids in numerical importance as vectors of virus diseases. The following are a few examples of destructive virus diseases that are transmitted by the leaf hoppers indicated: curly top of sugar beet by *Eutettix tenellus* Baker; what appears to be the same or a similar disease in Argentina by *Agallia stictocollis* Stål.; aster yellows by *Macrostes divisa* (Uhl.) and, to a lesser extent, by *Thamnotettix montanus* Van D. and *T. germinatus* Van D.; streak of maize by *Cicadulina mbila* Naude, *C. zeae*, and *C. storeyi*; cranberry false blossom by *Euscelis striatulus* Fall.; dwarf of rice by *Nephotettix apicalis* Motsch.; and peach yellows by *Macropsis trimaculata* Fitch. It will be noted that the vectors mentioned show a high degree of specificity for the diseases transmitted. In practically all cases, no other insect vectors are known for the particular virus transmitted although many have been tested. It is significant, also, that the vectors are rarely concerned in the transmission of any other virus although their host plants are often subject to several different virus diseases, and the host range of the virus transmitted is often very wide.

The plant hoppers, or lantern flies, include several important vectors. *Peregrinus maidis* Ashm. was reported by Kunkel (1922) as a vector of corn mosaic in Hawaii. Ocfemia (1934) and Mungomery and Bell (1933) have shown that the Fiji disease of sugar cane is transmitted by the two fulgorids (*Perkinsiella vastatrix* Breddin and *P. saccharicida* Kirk).

White flies have been shown to transmit a few virus diseases. Leaf crinkle, or leaf curl, of cotton in Nigeria and Sudan is transmitted by *Bemisia gossypiperda* Misra and Lamba (Golding 1930, and Kirkpatrick 1930, 1931). The disease is not transmitted by artificial inoculation, and the insect transmission appears to be biological and specific although Paoli (1931) claims that in Italian Somaliland it is transmitted by a leaf hopper (*Empoasca faciulis* Jac.). A similar disease of tobacco in Southern Rhodesia, probably caused by the same virus, is reported by Storey (1932b) as spread by *B. gossypiperda*. The host range of the virus is not definitely known. Cassava mosaic is said to be transmitted in Africa by *B. nigeriensis* Corb. (Golding

1936) and by *B. gossypiperda* var. *mosaicivectura* (Lefevre 1935, Storey and Nichols 1938).

The *scale insects* and *mealy bugs* are common sucking insects and affect a wide variety of plants, but they have been shown to transmit relatively few virus diseases. Elmer (1925) and Fajarado (1930) have claimed that mealy bugs are vectors of bean mosaic, and Olitsky (1925) reports *Pseudococcus citri* as a vector of tomato and tobacco mosaic. These reports, however, need further study and confirmation.

4. *Hemiptera*.—The insects of this order also have piercing-sucking mouth parts, and although many of them are plant feeders only relatively few are known to transmit virus diseases of plants. A lace bug of the family Tingidae, *Piesma* (*Zosmenus*) *quadrata* Fieb., transmits the crinkle-leaf disease of sugar beet in Europe (Wille 1928, 1929) and “savoy,” a similar although different disease of sugar beets in the United States, recently (Coons, Kotila, and Stewart 1937) has been shown to be spread by *P. cinerea*.

A few of the plant bugs have been incriminated in the transmission of certain virus diseases that are easily transmitted by mechanical means. Most of them belong to the family Miridae. For example, the tarnished plant bug (*Lygus pratensis* L.) transmits the spindle tuber of potatoes (Goss 1928), potato mosaic (Elze 1927), and spinach blight (McClintock and Smith 1918). It is probable that such transmission is purely mechanical and incidental. Smith (1934) has pointed out that this family of insects, although composing one of the largest groups of plant-feeding insects, is relatively insignificant as vectors of the viruses. He believes that the toxicity of the insect's saliva for the plant tissues in the immediate vicinity of the feeding puncture is responsible for their inefficiency as vectors of virus diseases. The zone of dead cells surrounding the feeding wound apparently keeps the virus from coming in contact with the protoplasm of the adjacent living cells. The possibility of the saliva being toxic to the virus is also recognized, but there is little evidence available to support the theory.

5. *Coleoptera*.—Like the Orthoptera, the members of this order, the beetles and weevils, are all chewing insects and play a relatively small part in virus transmission. About a half-dozen species have been incriminated, including the cucumber beetles

[*Diabrotica vittata* Fabr. and *D. duodecimpunctata* (Fabr.)] and the Colorado potato beetle (*Leptinotarsa decimlineata* Say), which are reported to transmit the viruses of cucurbit mosaic and spindle tuber of potatoes, respectively. Both these viruses are transmitted readily by artificial sap inoculation, and the beetles in all probability transmit the virus in a mechanical manner.

It will be seen from the foregoing that the sucking insects as a group are much more effective as vectors of virus diseases than the chewing insects. All sucking insects, however, are not equally effective. Some that feed on virus-infested plants appear to be unable to transmit the virus or else transmit it mechanically and incidentally. Others transmit viruses so effectively and with such regularity that it is evident that the transmission is the result of a highly specialized adaptation.

METHODS OF VIRUS TRANSMISSION BY INSECTS

If we consider the methods of virus transmission in the light of the system of classification given in Chap. IV, it will be seen that they practically all fall in class 3, "Dissemination with wounding." Insect transmission of plant viruses without wounding of the plant tissues is not known. There are examples of purely mechanical transmission and examples of biological transmission. There are examples of biological transmission in which the insect is obligatory and also those in which the insect is incidental.

Mechanical transmission is the usual method of those vectors with chewing mouth parts, such as the grasshoppers and beetles. Biological transmission is found only in the sucking insects, but transmission by sucking insects is not always biological. It has been shown that many sucking insects transmit viruses mechanically and that certain aphids, such as *Myzus persicae*, which transmit certain viruses biologically, may transmit others in a purely mechanical manner. Kunkel (1935) listed 25 insects that were believed to transmit viruses biologically. The list has increased since then and includes approximately 35 species, composed of about 20 leaf hoppers (including the fulgorids), 10 or more aphids, 2 or 3 white flies, and 3 or more species of thrips. The nature of biological transmission of viruses by insects is not clearly understood, and the term "biological

transmission" is used rather loosely. Biological transmission of plant viruses by insects usually has one or more of the following attributes:

1. An apparent multiplication or increase of the virus in the insect's body.
2. An incubation period in the body of the insect, that is, a necessary period after feeding on infected plants before the insect becomes infective or viruliferous.
3. A degree of specificity between the insect and the virus that it transmits.
4. An obligatory relationship.
5. A relation between the age or life stage of the insect and its ability to transmit the virus.
6. Congenital transmission of the virus from one generation to the next.

Each of these attributes must be considered in more detail if we are to have a clear picture of what is known about the biological relationship between insect and virus. The nature of this relationship has occupied the attention of numerous investigators during the past few years, and although there are many conflicting viewpoints, much progress has been made. A critical evaluation of some of the most important investigations in the field has been published recently by Storey (1939).

MULTIPLICATION OF THE VIRUS IN THE INSECT BODY

The evidence for multiplication or increase of the virus in the body of the insect vector is largely circumstantial and is based chiefly on the fact that the virus is known to persist in the insect's body for a relatively long time. Few quantitative measurements have been made, and the evidence is somewhat conflicting. It has been shown (Kunkel 1926) that the virus of aster yellows may persist in the body of a viruliferous leaf-hopper vector kept on nonsusceptible plants for the length of its natural life, which may be as long as 100 days. In other insects, where transmission is purely mechanical, the virus disappears from the vector in a short time after feeding on an infected plant, and the insect becomes noninfective. From this it is concluded that the persistence of the virus for long periods of time is to be explained by assuming a multiplication in the insect's body. It seems reasonable to expect that the small amount of the virus taken up in a short period of feeding on an infected plant would be lost after feeding for such a long time on nonsusceptible plants unless there were some multiplication

in the insect body. The chief objection to this assumption lies in the fact that the so-called "nonsusceptible" plants on which insects are fed are not completely immune and that a renewed supply of virus may be obtained from them. However, the virus is known to multiply in contact with living plant cells, and it seems reasonable that it could also multiply in the tissues of insect. The fact that it disappears so quickly from some insects and survives so long in others suggests the possibility of a multiplication of the virus in the latter.

Further circumstantial evidence of multiplication of the virus of aster yellows in its vector is found in the fact, demonstrated by Kunkel (1937), that the virus may be inactivated in the insect's body by exposure to temperatures of 31 to 32 degrees for a sufficiently long time; but that if the exposure is of short duration, the insect only temporarily loses its ability to transmit the virus. The spontaneous regaining of the virus is interpreted by Kunkel as a multiplication of the small quantity of the virus not destroyed by the shorter heat treatment.

Stronger evidence has been presented by Fukushi (1933, 1935, 1939), who has demonstrated that the virus of the dwarf disease of rice is congenitally transmitted through the eggs of its vector (*Nephotettix apicalis*). He demonstrated transmission of the virus to the seventh generation. A single female vector infected 38 plants consecutively in 24-hour feeding periods, and the progeny of 15 eggs deposited by this female infected 201 plants without access to new sources of the virus. In one experiment, the progeny of one viruliferous egg, consisting of 26 leaf hoppers of five generations, inoculated more than 1,000 rice plants. Fukushi concludes that, since the amount of the virus originally contained in an egg or in the body of a nymph must be extremely minute, it seems necessary to postulate the multiplication of the infective principle within the insect.

On the other hand, Freitag (1936) and Bennett and Wallace (1938) have presented evidence which leads them to conclude that the curly-top virus does not multiply in the body of *Eutettix tenellus*. These workers have shown by quantitative measurements that the virus content of viruliferous leaf hoppers decreases with the time elapsing after feeding on infected beets and that the time of survival in the vector decreased as the time of previous feeding on infected beets was decreased. Viruliferous

vectors that had become noninfective by long confinement on immune plants regained their infectivity when fed again on infected beets. There was no evidence of concentration of the virus in the salivary glands, and the evidence pointed to the blood, as the principal reservoir of the virus in the insect body.

Evidence for the multiplication of animal viruses in their insect vectors is also somewhat contradictory. According to Davis, Frobisher, and Lloyd (1933), the virus of yellow fever does not increase in the body of *Aedes aegypti*. Quantitative measurements of the virus were made by diluting the crushed bodies of mosquitoes until no infection was obtained, when the diluted suspension was inoculated into susceptible animals. In contrast to this, Merrill and TenBroeck (1934, 1935) showed that the virus of equine encephalomyelitis would multiply in the bodies of *A. aegypti*. These workers artificially inoculated mosquitoes with the virus through 17 successive transfers, and in the last mosquito the concentration of the virus equalled that found in the first. These facts seem to justify the conclusion that this virus multiplies in the body of its insect vector. It appears that all viruses do not behave alike in regard to this phenomenon.

Quantitative measurement of the virus in the bodies of insects is difficult and often may not be reliable when the technique involves the inoculation of plants with the juices of crushed insects. Black (1939) has shown that virus activity is often inhibited by insect juices. The juices of several species of aphid and leaf hopper effectively inhibited the infectivity of tobacco mosaic, and the juices of macerated clover leaf hoppers inhibited that of six other viruses. It was shown that the juices did not destroy the virus, because by suitable methods it could be separated in active form from the inhibiting juices. It was concluded that the inhibitory action was caused by action on the inoculated plant rather than on the virus itself.

THE INCUBATION PERIOD IN THE INSECT BODY

It is a well-recognized fact that certain vectors of virus diseases do not become viruliferous until a certain time has elapsed after feeding upon an infected plant. This period of delay is usually called an "incubation" or a "latent" period although very little is known about the nature and cause of the delay.

The incubation period should not be confused with the so-called "threshold" period, which is the shortest length of time the vector must feed upon the infected plant in order to secure virus enough to become viruliferous. The threshold period has been determined for only a few species. Storey (1938) has determined it for *Cicadulina mbila* and the streak virus and found it to be approximately 5 minutes. For *Eutettix tenellus* and the curly-top virus it may be as short as 1 minute, according to Bennett and Wallace (1938).

The length of the incubation period varies with different insects and diseases from 20 minutes to 10 days or more and is modified by many influencing factors. The incubation period of the beet curly-top virus in the beet leaf hopper may be as short as 20 minutes when a large number of hoppers are used in the experiment. The shortest period obtained for a single leaf hopper is 7 hours (Severin 1921). The minimum incubation period in the experiments of Bennett and Wallace (1938) was 4 hours. Swezy thinks that periods as short as 20 minutes or even 1 hour are not normal and can be explained by abnormal body structure which obstructs passage of the food and virus and causes its ejection or regurgitation shortly after it has been ingested. The possibility of an abnormal and unusually rapid passage of the virus through the body into the salivary glands is also recognized.

The minimum incubation period for the virus of aster yellows in the adult leaf hopper, according to Kunkel (1926), is 10 days. A longer period is required in nymphs. Similar incubation periods have been shown for a number of other insect vectors, including *Cicadulina mbila* and the streak disease of maize (*Myzus persicae*), potato leaf roll (*Thrips tabaci*), and the yellow spot of pineapple. The length of the incubation period apparently is influenced by temperature; the higher the temperature, within limits, the shorter the period (Storey 1928).

Several theories have been offered to explain the necessity for an incubation period. It has been suggested by Smith (1934) that the virus particles may be absorbed on the surface of the tissues of the insect's mouth parts and that the insect becomes infective only after the absorbing surfaces have become saturated. This, however, would not explain the incubation period when the

insect feeds on infected plants for a short period and is then transferred to nonsusceptible plants.

The most probable explanation is based upon the theory that the virus particles must pass through the walls of the intestinal tract into the blood and from there into the salivary glands before they can be introduced into another plant. This hypothesis is supported by the fact that the insects in which an incubation period occurs (Homoptera and Hemiptera) are equipped with an esophageal valve that provides a one-way passage of the plant sap and prevents regurgitation.

Swezy (1930) and Smith (1931a) have traced the course of dyes through leaf hoppers and aphids, respectively, and have shown that the dyes reach the salivary glands in a period of time shorter than the incubation period of the virus concerned. This does not invalidate the hypothesis, for the rate of diffusion of the virus particles may be much slower than the dyes used. Stanley (1937a) and others have shown recently that the protein molecule composing the virus of tobacco mosaic has a molecular weight greater than that of any other known protein, and filtration experiments have indicated that the particles of tobacco mosaic virus are among the smaller virus particles. Proteins of such high molecular weight would probably diffuse more slowly than dye particles.

Storey (1928, 1932a, 1933) has thrown some light on the course of the virus in the insect by his studies on *C. mbila* and the virus of maize streak. Storey discovered a strain of the vector that failed to become infective after feeding on infected plants. He showed that this failure was caused by the impermeability to the virus of the wall of the intestinal tract. When the intestinal wall was punctured with a fine needle after the insects had fed upon an infected plant, those insects which recovered were infective. Storey also was able to demonstrate the presence of the virus in the blood of normal insects after feeding on diseased plants as early as 5 to 6 hours before the insect became infective. It is highly probable that the normal course of the virus is from the digestive tract of the insect into the blood by diffusion, from the blood into the salivary glands, and from the salivary glands into the plant. An incubation period based on the time required for completing this course would more

nearly explain the known facts than any other hypothesis presented. This is about the only way an incubation period could be explained in those cases where the virus may be artificially transmitted by sap inoculation.

Hamilton (1935) has attacked the problem in an interesting manner by feeding aphids (*M. persicae*) on agar containing polonium, a radioactive substance, that could be traced by measurement with an electroscope. It was shown that the insect picks up the polonium and transmits it in constant amounts to leaves on which it subsequently feeds. It is concluded that the polonium, and in all probability the virus also, is transmitted through the insect's body and not mechanically on the stylets. The same author (Watson 1936) has made a careful study of the factors affecting the transmission of a virus designated as Hy III by *M. persicae* Sulz. The virus, which is readily transmitted by artificial sap inoculation, appears also to be transmitted biologically by the aphid. The amount of transmission obtained varied with changes of external conditions as well as with conditions within the insect. Of particular interest was the fact that when the insects were starved for a few hours before feeding a greater efficiency in transmission was obtained with feeding periods of 2 to 5 minutes on infected plants than with feeding periods of longer duration. This apparently was not true if the aphids were not previously starved. Two possible hypotheses were offered to explain these results. The first is based on the assumption that some of the virus is digested by the insect and that "starvation may cause the glandular cells of the stomach wall to enter a resting phase, so that the digestive enzymes are not secreted until a short time after food has entered the mid-gut."

The second hypothesis assumes the production of antibodies to the virus within the insect's body which, after a short time, confers partial immunity against the virus.

The existence of a developmental cycle in the virus comparable to that found in certain parasitic protozoa transmitted by insects has been suggested by many workers, but there is no evidence whatever to support it. The conclusion of Stanley (1937a) that viruses are in reality proteins of high molecular weight, if confirmed, would seem to preclude the possibility of any such cyclic change.

The latent period of the virus in the plant appears to vary with the number of insects used for inoculation, the shorter latent periods usually being obtained when a relatively large number of vectors are used. Severin (1931) and Carsner and Lackey (1929) have explained this by a theory of mass action which holds that, among other things, the length of the latent period varies in indirect proportion to the amount of inoculum introduced or to the number of vectors used in inoculation. This implies a cumulative effect of many small doses of virus injected by many different vectors. Storey (1938), on the other hand, feels that the evidence does not justify this conclusion, claiming that each separate inoculation is independent of all others and that several subinfective doses of a virus cannot unite in the plant to produce an infective dose.

SPECIFICITY OF VIRUS TRANSMISSION BY INSECT VECTORS

There are numerous well-demonstrated cases of specificity between insect vectors and the viruses they transmit. The most striking examples are found in those virus diseases transmitted by leaf hoppers, including beet curly top and *Eutettix tenellus*, peach yellows and *Macropsis trimaculata*, cranberry false blossom and *Euscelis striatulus*, etc. As other examples of specific relationships may be mentioned leaf curl of cotton transmitted by *Bemisia gossypiperda* and pineapple yellow spot transmitted by *Thrips tabaci*. One should remember, however, that the evidence for specificity of transmission is generally of a negative character and that there is always the possibility of additional vectors being discovered.

Although transmission is biological and there is a certain amount of specificity, the specificity is not always absolute, and transmission of a given disease is not always confined to one species of insect. Aster yellows, for many years thought to be transmitted only by *Macrostelus divisa*, is now known to be transmitted by three different species of leaf hopper (Severin 1934b). Spotted wilt of tomato is transmitted by two species of thrips (Smith 1931b, Samuel and Bald 1931), rice dwarf by two species of leaf hopper, maize streak by three species of leaf hopper; and potato leaf roll is transmitted by at least six species of aphid. These viruses are all biologically transmitted, and each one is transmitted by closely related species of sucking insect.

Group specificity in transmission is the most common condition among the aphid vectors.

There are also insects that biologically transmit two distinct virus diseases, yet show a high degree of specificity for the two diseases and appear to be unable to transmit other viruses that affect their host plants. As examples may be mentioned *M. dirisa*, which transmits two distinct strains of aster yellows; *Myzus persicae*, which transmits biologically potato leaf roll and also dahlia mosaic (Brierly 1933); and *Aphis gossypii*, which is a vector of both "yellow flat" and mosaic of lilies.

Specificity is manifested by certain insects that transmit selectively one virus from a plant affected with two or more distinct viruses. Hoggan (1929, 1930, 1931, 1933, 1934) has shown that, when *Myzus persicae*, *M. solani*, and *Macrosiphum gei* are fed on tobacco plants affected with both cucumber mosaic and tobacco mosaic, they will transmit only the virus of cucumber mosaic. This specificity may be based not on a biological relationship with the vectors but rather with the feeding habits of the insects on the tobacco plants, for they will readily transmit the tobacco virus from tomato plants. Smith (1931a) has reported selective transmission in which different viruses were consistently transmitted from the same compositely infected potato plant by insect and needle inoculations, respectively.

In contrast to the high degree of specificity shown by certain insects and viruses, it is known that the yellow dwarf of onions may be transmitted readily by more than 50 species of aphids as well as a number of other sucking insects. Likewise, a single aphid (*M. persicae* Sulz.) is known to transmit more than 20 different viruses.

The apparent specificity of certain groups of insects for viruses that produce similar symptoms has led to the use of insect relationships in the classification of viruses (Elze 1931b, Storey 1931, Johnson and Hoggan 1935, Smith 1934). According to Smith there are four more or less distinct groups as follows: (1) the mosaic group transmitted largely by aphids, (2) the yellows group transmitted by the leaf hoppers, (3) the group characterized by the production of spots with concentric rings, transmitted by thrips, and (4) those transmitted by the white flies and causing a thickening of the veins and abnormal leafy outgrowths on the lower side of the leaves. The extent to which insect relationships

TABLE I.—THE CLASSIFICATION OF PLANT VIRUSES¹

The plant viruses	Transmissible by sucking insects (A_1)	Not transmissible mechanically by plant extract (B_1)	Not transmissible by aphids (C_1)	Transmissible by leaf hoppers (D_1)	
				Not transmissible by leaf hoppers (D_2)	
		Transmissible mechanically by plant extract (B_2)	Transmissible by aphids (C_2)	Rosaceae susceptible (D_1)	
				Rosaceae not susceptible (D_2)	
			Longevity <i>in vitro</i> less than 7 days at 22°C. (C_1)	Thermal death point below 60°C., 10 min. (D_1)	
				Thermal death point 60°C., or above, 10 min. (D_2)	
	Not transmissible by sucking insects (A_2)	Transmissible mechanically by plant extract (B_1)	Longevity <i>in vitro</i> 7 days or more at 22°C. (C_2)	Thermal death point below 80°C., 10 min. (D_1)	
				Thermal death point 80°C., or above, 10 min. (D_2)	
			Not transmissible mechanically by plant extract (B_2)	Longevity <i>in vitro</i> 7 days or more at 22°C. (C_1)	Thermal death point 60°C., or above, 10 min. (D_1)
					Thermal death point less than 60°C., 10 min. (D_2)
				Longevity <i>in vitro</i> less than 7 days at 22°C. (C_2)	Thermal death point 60°C., or above, 10 min. (D_1)
					Thermal death point less than 60°C., 10 min. (D_2)
Not transmissible by sucking insects (A_2)	Not transmissible mechanically by plant extract (B_2)	Transmissible by grafting (C_1)	Potato susceptible (D_1)		
			Potato not susceptible (D_2)		
		Not transmissible by grafting so far as known (C_2)	Transmissible by other vegetative means (D_1)		
			Not transmissible by other vegetative means (D_2)		

¹ After Johnson and Hoggan (1935).

may be used in virus classification is shown in the scheme for classifying the major groups proposed by Johnson and Hoggan (1935) (see Table I).

The nature and cause of the specificity shown by certain insect vectors and the viruses they transmit have never been satisfactorily explained. A work of great significance in the study of this problem is that of Storey (1932a, 1933) in which it was shown that within a single species (*Cicadulina mbila* Naude) there are two strains or races, one of which is unable to transmit the virus of maize streak. These two races were designated as "inactive" and "active" according to their ability to transmit the virus. By breeding experiments, the ability to transmit the virus was shown to be inherited as a dominant sex-linked character. Thus when active females were crossed with inactive males, the F_1 progeny were all active while the F_2 generation yielded active females and both active and inactive males. When active males were crossed with inactive females, the F_1 generation contained active females and inactive males. The F_2 of this cross produced both active and inactive females. No morphological differences between the races were observed. Further experiments showed that both active and inactive insects ingested the virus, which could be detected also in the feces of each race. In the inactive race, the virus was unable to diffuse through the intestinal wall into the blood and consequently did not reach the salivary glands. When the intestinal walls of inactive insects were punctured with sharp needles after or just before feeding on infected plants, they became infective. The essential difference between the races appears to lie in the unequal permeability to the virus of the intestinal walls. This is a very significant discovery and suggests that specific selective permeability of the intestinal walls of the insect vectors may account for much of the specificity observed.

Individual differences in ability to transmit a virus have not been so well demonstrated for any other vector of plant viruses, but Huff (1931) has reported a similar condition in two species of mosquito (*Culex* spp.) in relation to *Plasmodium cathemerium*, the cause of bird malaria. The ability of these vectors to transmit the pathogen is inherited, according to Huff, as a simple Mendelian recessive factor.

Bennett and Wallace (1938) have shown that several species of insects which are not vectors of curly top of beet, including *M.*

persicae, *A. rumicis*, *Aceratogallis californiaca*, *Hercothrips femoralis*, and others, acquired the virus by feeding on infected beets and retained it for periods ranging from 1 to 21 days but were unable to transmit it to sugar beets. It was concluded that the inability of these insects to transmit the virus was caused by the presence of an effective barrier to virus passage in some part of the insect's body.

THE OBLIGATORY NATURE OF INSECT TRANSMISSION OF CERTAIN VIRUSES

When a virus can be transmitted only by one or two closely related insects and cannot be transmitted artificially (except by grafting), the insect transmission is considered obligatory. There are few, if any, viruses that cannot be transmitted by grafting. Failure to transmit a disease by grafting would be strong presumptive evidence that it is not caused by a virus. Obligatory insect transmission obviously must be based on negative evidence, and there is always the possibility that a suitable technique for artificial transmission will be discovered. Nevertheless, failure of all recognized methods of transmitting the virus, except by grafting and through the agency of specific insect vectors, is considered good evidence that transmission is not mechanical but that a biological relationship is concerned. The following virus diseases are good examples of those which have not been transmitted artificially except by grafting, and the insect vectors indicated are considered essential for the spread of the disease in nature. The list, of course, is subject to modification by new evidence.

1. Aster yellows [*Macrosteles divisa* (Uh.), *Thamnotettix montanus* Van D., and *T. germinatus* Van D.].
2. Dwarf of rice (*Nephotettix apicalis* Motsch. and *Deltocephalis dorsalis* Motsch.).
3. Peach yellows (*Macropsis trimaculata* Fitch).
4. Cranberry false blossom (*Euscelis striatulus* Fall.).
5. Streak of corn [*Cicadulina mbila* (China), *C. storeyi* China, and *C. zeae* China].
6. Fiji disease of sugar cane (*Perkinsiella vastatrix* Breddin and *P. saccharicida* Kirk.).
7. Cotton leaf curl (*Bemisia gossypiperda* Misra and Lamba.).
8. Leaf curl of sugar beet (*Piesma quadrata* Fieb.).

Johnson and Hoggan (1935) list 28 viruses that are transmitted specifically by sucking insects and are not transmissible mechan-

ically by plant extracts, and there have been several additions since the tabulation was made.

THE RELATION OF THE AGE OR LIFE STAGE OF THE INSECT VECTOR AND ITS ABILITY TO TRANSMIT THE VIRUS

Another indication of biological transmission of viruses is offered by certain vectors that are unable to transmit the virus in the nymphal or larval stages. Kunkel (1926) showed that nymphs of *Macrosteles divisa* (Uhl.) often were unable to transmit the virus of aster yellows. This was explained by pointing out that the minimum incubation period of the insect was longer than the period of metamorphosis.

A somewhat different relationship exists between the thrips (*Thrips tabaci* and *Frankliniella lycopersici*) and the viruses they transmit. Bald and Samuel (1931) have shown that, in order to transmit the virus of the spotted wilt of tomato, *F. lycopersici* must have fed in the larval stage on infected plants. The adults appear unable to pick up the virus by feeding. Linford (1932) showed the same relationship for *T. tabaci* and the yellow spot of pineapple. Smith (1932) confirmed this peculiar relationship in transmitting the spotted wilt of tomato with *T. tabaci*.

The relationship here is more difficult to explain than that between *M. divisa* and the aster-yellows virus because the thrips live in the larval stage much longer than the incubation period, which is rarely longer than 10 days. It is still more difficult to explain when we recall that the virus of spotted wilt may be transmitted mechanically by sap inoculation. The explanation in all probability must be sought in the anatomy or physiology of the digestive system of the insects.

CONGENITAL TRANSMISSION OF VIRUS IN THE INSECT VECTOR

Despite the intricate biological relationships between viruses and their insect vectors, congenital transmission of viruses appears to be the exception rather than the rule. As early as 1918, McClintock and Smith reported that the virus of spinach blight was transmitted from infective aphids (*Myzus persicae* and *Macrosiphum gei*) to their viviparously produced young through four generations. These conclusions were based on the assumption that the host on which the young were reared was

immune to the virus, an assumption later proved to be erroneous. Hoggan (1933) and others have been able to show that such congenital transmission does not occur in these aphids. Similar proof of the noninheritance of the virus of potato leaf roll by aphids has been obtained by Elze (1927) and Smith (1929).

Many leaf-hopper vectors do not transmit the virus to their offspring. Bennett and Wallace (1938) have shown that the virus of curly top is not transmitted through the eggs. Similar conclusions were reached by Kunkel (1926) in regard to the aster-yellows virus and *Macrostelus divisa* and by Storey (1928) for maize streak and *Cicadulina mbila*.

However, in 1933 and 1939 Fukushi presented evidence to show that the virus of the dwarf disease (stunt) of rice is congenitally transmitted through the eggs of the vector (*Nephotettix apicalis* Motsch. var. *cincticeps* Uhl.). Insects of known history, males and females in pairs, some infective and some noninfective, were placed on healthy rice plants enclosed in glass tubes where they were allowed to remain only 1 day, being transferred to new healthy plants each day. Eggs were deposited in the lower part of the leaf sheath. As the young nymphs emerged from the eggs, they were transferred individually to healthy plants before they had an opportunity to feed on the plants in which the eggs were deposited. They were kept on these plants for 2 months or longer.

The results indicated that the virus was transmitted through the eggs to the nymphs. When infective females were paired with infective males, the majority of the offspring in all progenies was infective. When infective females were paired with noninfective males, both infective and noninfective progenies were found; but when noninfective females were paired with infective males, none of the progenies was infective.

Fukushi states that when eggs were deposited in infected plants by noninfective females none of the progeny was infective. This is added evidence that the eggs become infected in the early stages of development in the ovary. Since some individuals of the progenies from infective females were noninfective, it was concluded that some of the ovaries may escape infection. Because of the significance and striking nature of these results the tabulated summary of Fukushi's experiments is given in Table II.

THE EFFECT OF THE VIRUS ON THE INSECT VECTOR

In view of the close biological relationship shown by certain viruses and their insect vectors, one naturally is interested in knowing what effect the virus has upon the insect. Is the insect infected with a virus disease like the plant? This is perhaps only

TABLE II.—CONGENITAL TRANSMISSION OF VIRUS OF RICE DWARF IN THE LEAF HOPPER (*Nephotettix apicalis*)*

Parents		No. of progeny tested	No. of viruliferous progeny
Female	Male		
Infective female × infective male			
e279-4-3(♀)	e279-4-4(♂)	7	5
e'34(♀)	e'29(♂)	14	13
e'39(♀)	e'36(♂)	8	8
e'49(♀)	e'68(♂)	4	4
e'53(♀)	e'58(♂)	1	1
Infective female × noninfective male			
e279(♀)	a(♂)	13	13
e151-4(♀)	b(♂)	16	15
e151-1(♀)	c(♂)	26	17
e151-4-4(♀)	d(♂)	6	0
e279-4(♀)	e(♂)	35	33
e279-1(♀)	f(♂)	27	0
e151-1-16(♀)	g(♂)	30	0
e'51(♀)	h(♂)	8	8
e279-4-5-3-3(♀)	i(♂)	5	5
e'62(♀)	j(♂)	2	2
e'69(♀)	k(♂)	2	0
e'65(♀)	l(♂)	1	1
e'61(♀)	m(♂)	12	6
Noninfective female × infective male			
a(♀)	e151-1-22(♂)	9	0
b(♀)	e151-1-13(♂)	19	0
c(♀)	e'151-1-13(♂)	5	0
d(♀)	e'47(♂)	9	0
e(♀)	e279-4-32(♂)	14	0
f(♀)	e'64(♂)	4	0
g(♀)	e'72(♂)	3	0

* After Fukushi. ♂ and ♀ denote infective male and female, respectively, and ♂ and ♀ stand for noninfective male and female.

an academic question and may depend entirely upon our definition of a disease. No one has ever observed a detrimental effect

of plant viruses upon the insect vectors. There are several very destructive virus diseases of insects, and it is obvious that the effect of the plant viruses on the insects is in no way comparable to these. Dobrosky (1931) was unable to find by histological methods any lesion or any visible effect of the virus of aster yellows on the leaf-hopper vector. Smith (1934) stated that he, too, was unable to find any visible effect of two potato viruses on aphids. Blattny (1931), on the other hand, found that the areola around the nucleus of the cells of the salivary glands was darker in infective aphids than in noninfective ones. Hartzell (1937) reports the presence of intracellular inclusions in the cells of the intestinal wall and salivary glands of viruliferous specimens of *Macropsis trimaculata*, the vector of peach yellows. These bodies were similar to those observed in the cells of infected peach tissue, and they were not found in healthy peach tissue or in nonviruliferous leaf hoppers. Similar bodies were observed in viruliferous specimens of *Macrosteles divisa* and in asters affected with yellows. The observations were made on living tissues and unstained fixed material. This is the first report of inclusion bodies in the insect vectors of plant viruses. It shows that the virus is in intimate contact with the protoplasm of the cells of the insects but does not necessarily mean that the insects are affected with a virus disease. Whatever may be the significance of the cell inclusions, it is probably true that the viruses are less injurious to the insect vectors than to plants.

It has been observed that the sucking insects, which include the specific vectors, are all provided with a mycetome. It often has been suggested that the mycetome or its symbiotic micro-organism may be in some way related to the viruses transmitted by the insect. However, no convincing evidence of such a relationship has been presented. Carter (1939) failed to find any correlation between the presence of symbiotes in *Cicadulina mbila* and their ability to transmit the virus of maize streak.

Stanley (1937a) has shown that one of the chief effects of the tobacco-mosaic virus on the tobacco plant is to dominate protein metabolism so that abnormally large amounts of a foreign protein (the virus) are synthesized. Since there is some evidence that the virus multiplies in the insect vectors, it is likely that the virus modifies the nitrogen metabolism of the insect. It is difficult to determine to what extent this effect is injurious. It

is entirely possible that the virus in some way may be beneficial to the insect vector. Stanley (1937b) and Bawden and Pirie (1937) have concluded that the soluble-protein content of tobacco plants affected with tobacco mosaic is considerably higher than that of healthy plants. Although this conclusion has been questioned by Martin, Balls, and McKinney (1938) and the total protein is not increased by all viruses in all plants, it is agreed that the nitrogen metabolism of the diseased plant is greatly altered. It is not known in what form the modified protein occurs, but apparently much of it is the actual virus protein. It is well known that the protein supply is the limiting factor in the nutrition of insects which feed on the sap of plants and that they must ingest very large quantities of water and carbohydrates in order to get enough nitrogenous food for normal nutrition. Mumford and Hey (1930) have pointed out that a high nitrogen content of plants stimulates reproduction in insects which feed upon them and in general makes the plant more susceptible to insect attacks. A higher protein content or a higher percentage of a more suitable protein in available form would be of decided benefit to the sucking insects. It is not yet known whether insects are able to utilize the virus proteins as food or whether the virus proteins in any way influence their metabolism. Watson (1936), in an effort to explain certain facts concerning the transmission of a virus by *Myzus persicae*, has suggested that some of the virus may be digested by the insects and that only a part of the ingested virus is needed for effective transmission. Feeding experiments with the various vectors and purified virus proteins should throw light on some of the obscure relationships between insect and vector.

Complicated structures or complicated adaptations rarely occur in nature without serving some important function. They usually arise through natural selection on the basis of their survival values. One is therefore inclined to look for the possible benefits derived from the association by each of the associated entities. Numerous cases have been cited of the evolution of an association of insects and microorganisms for mutual benefit. There is no convincing reason why association of insects and viruses may not have arisen in a similar manner. It requires very little imagination to see such a beneficial association between the leaf hopper (*Eutettix tenellus*) and the curly-top virus. According

to Carter (1930), this insect is very susceptible to high humidity and thrives best in arid regions in relatively thin stands of its host plants. It will not thrive in heavy luxuriant stands of weeds or sugar beets. If sugar beets are well developed in a good stand when the leaf hoppers migrate to the beet fields, the hoppers do not multiply rapidly on account of the high relative humidity and do not build up large populations. On the other hand, if the hoppers migrate to the field when the plants are small, the unfavorable humidity of dense stands is precluded by infecting the plants with the curly-top virus and preventing their normal development. If the leaf hoppers were not vectors of the curly-top virus, the sugar beets would grow normally and in all probability would not be a favored host of the leaf hopper.

The question of whether a virus-infected plant is a more favorable host than a healthy plant for the insect vector has been given very little attention in virus studies. It is of interest, therefore, that Carter (1939a) in a recent study of populations of *Thrips tabaci*, with reference to transmission of pineapple yellow spot, has found that the populations on diseased weeds (*Emilia sonchifolia*) are consistently higher than those on healthy plants. It is concluded that infected plants are more suitable hosts for the vector because, as a result of delayed maturity, they persist for a longer time than healthy plants, and because the excessively curled leaves afford valuable shelter for the vector.

The benefits, if any, derived by the vectors of other virus diseases are not so evident. The dependence of the virus upon the insects for transmission is obvious and leaves no doubt of the benefits derived from the association by the virus. Whatever the significance of the association may be, there is yet much to learn about the relationships between the viruses and their vectors. The problem offers a fascinating field of research, and its solution should be of fundamental significance in the fields of both plant and animal pathology.

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CHAPTER IX

INSECTS AND VIRUS DISEASES (*Continued*)

SELECTED EXAMPLES OF VIRUS DISEASES TRANSMITTED BY INSECTS

The number of virus diseases of plants known to be transmitted by insects is so very large that it is impractical to discuss them all in detail. Smith (1937*a*) lists a total of 136 viruses, excluding the numerous strains of those given numerical rank, and nearly all of them are transmitted by one or more species of insects. Holmes (1939) has applied Latin binomials to 89 species and 30 varieties. Although there is wide variation in the relationships between the viruses and their insect vectors, the principles involved in transmission are essentially the same for many of the virus diseases. Moreover, the information about the methods of insect transmission is meager for some of them. Therefore, only a few of the better known virus diseases, selected to illustrate representative types of transmission, will be discussed, and these will be treated primarily from the standpoint of transmission phenomena.

An effort has been made to arrange the diseases in groups according to the methods by which they are transmitted in nature, with particular reference to insect vectors. For a number of obvious reasons, it is not possible to draw sharp lines between the different groups. It is believed, however, that the arrangement followed will aid in visualizing the important features of insect transmission of the virus diseases.

1. TRANSMISSION BY MECHANICAL SAP INOCULATION; INSECT TRANSMISSION PURELY MECHANICAL

Potato Spindle Tuber (*Solanum Virus 12).**—The name "spindle tuber" was applied to this disease by Martin in 1922. Its infectious nature was established first by Schultz and Folsom (1923) who showed that it could be transmitted by aphids and

* Classification of Kenneth Smith (1937*a*).

also by artificial sap inoculation. It is selected as an example of a highly infectious virus transmitted mechanically and non-specifically by many different insects. Spindle tuber has been found to be distributed generally throughout the potato-growing regions of the United States; according to Smith (1934), it is not known in Europe. Because of the rapidity with which the disease spreads in nature and the difficulty of detecting infected plants in early stages of development, spindle tuber is one of the most difficult to control of all the virus diseases affecting potatoes.



FIG. 135.—Potato spindle tuber. The tubers on the left are healthy; those on the right are affected with spindle tuber.

The most characteristic symptom of this disease, as indicated by the name, is the tendency of tubers to become excessively elongate, forming a spindle-shaped tuber tapering most at the bud end (Fig. 135). The color of the tubers of red varieties become less intense; the eyes at the apex are shallow and more numerous, with protruding "eyebrows." The yield of marketable tubers is always greatly reduced.

The symptoms of spindle tuber have been confused with those of unmottled curly dwarf, a disease very similar in many respects but thought by some workers to be caused by a distinct virus. The most extensive comparative study of the two diseases has been made by Goss (1930). Both diseases apparently have a narrow host range, as interspecific inoculations have failed to transmit them to other solanaceous species.

Transmission of Spindle Tuber.—Spindle tuber is highly infectious and is readily transmitted by many different agencies. It is easily transmitted by various methods of artificial sap inoculation. Goss (1931) has shown that spindle tuber, as well as unmottled curly dwarf, is transmitted readily by contact of cut seed pieces and by the cutting knife, as many as four successive seed pieces being successfully inoculated with the knife following its use in cutting a diseased tuber.

As would be expected with such a highly infectious disease, it is transmitted readily by many different insects. Goss (1931) tested the insects that were most prevalent on potatoes in Nebraska and succeeded in demonstrating transmission by the following: grasshoppers (*Melanoplus spp.*), flea beetles (*Epitrix cucumerus* Harris and *Systema taeniata* Say), the leaf beetle [*Disonycha triangularis* (Day)], the tarnished plant bug (*Lygus pratensis* L.), and the larvae of the Colorado potato beetle (*Leptinotarsa decimlineata* Say). A small number of trials with the leaf hopper (*Euscelis exitiosus*) gave negative results. Transmission by aphids (*Macrosiphum gei* and *Myzus persicae*) had previously been demonstrated by Schultz and Folsom (1923). Unmottled curly dwarf was transmitted by the same species of insects with the exception of the leaf beetle (*D. triangularis*).

The results of these experiments indicate strongly that insect transmission is entirely mechanical and that almost any insect that feeds on the potato and moves from plant to plant is a potential vector of the virus. In all probability, this fact accounts for the very rapid spread of spindle tuber under field conditions. Brentzel (1935) has reported an experiment showing the rapidity of spread of spindle tuber in North Dakota. Beginning with selected healthy seed stock, 1,000 tubers, selected at random, were replanted for 3 successive years. No precautions were taken to prevent the spread of the disease. After 3 years, 95 per cent of the plants were definitely affected with the disease. Goss (1929) reported a spread of 42.4 per cent by spindle tuber in Nebraska after 4 years, as compared with 9.7 per cent spread by leaf roll. Mild mosaic and rugose mosaic spread more slowly than leaf roll. In one year, when insects were more abundant, there was an increase in spindle tuber infection from 14 to 42 per cent.

Control of spindle tuber is relatively difficult and obviously depends upon rigid inspection of seed stock, early recognition of

EARTHWORMS

Earthworms and Clubroot of Crucifers.—Gleisberg (1922) has reported evidence to show that earthworms are concerned in the spread of clubroot of crucifers, a disease caused by a fungus (*Plasmodiophora brassicae* Woronin). Large irregular swellings develop on the roots of affected plants. Spores are formed within the cells of affected tissue and are liberated only after the plant dies and the roots decay. The spores germinate in the soil and form myxamoebae that find their way to the roots of new plants which they infect through the root hairs. Although infested areas of soil do not increase in size rapidly, the rate of spread is often faster than can be explained by the direct migration of the fungus in the soil.

Gleisberg placed 3 to 10 earthworms in pots of infested soil and allowed them to remain for 1 month, then removed them to new pots of disease-free soil which he planted to cabbage. Sixty per cent of the plants were infected in these pots, whereas none were infected in similar pots of noninfested soil to which no earthworms were added. The pathogen, in virulent form, was found to be abundant in the excreta of the worms, and it was concluded that earthworms in nature would carry the spores of *P. brassicae* for some distance in the soil. It was shown that deep plowing to turn under the infested soil is of only temporary value, because the earthworms soon bring enough of the inoculum to the upper layers to reinfest it.

The dissemination of *Plasmodiophora* in this way would be relatively local, because earthworms do not travel for very great distances. There is a possibility that they may be indirectly responsible for long-distance transmission by birds that feed upon earthworms. There is no experimental evidence, however, to show that this ever occurs.

SLUGS

On several occasions, slugs have been reported as agents of dissemination of fungus spores or bacterial plant pathogens, but as a rule they are of relatively little importance as vectors of plant diseases. As early as 1896, Wagner reported experiments with slugs (*Helix hortensis* Müll., *Succinia putris* L., and *Arion subfuscus* Drap.) showing that they effectively dissemi-

be transmitted by aphids (Allard 1917), these insects apparently are not important vectors of this particular virus. The tobacco plant is not a favorable host for aphids, and Hoggan (1930, 1931, 1934) has shown that *Myzus persicae*, *M. pseudosolani*, *M. circumflexus*, and *Macrosiphum solanifolii*, the most common aphids on tobacco, do not transmit this virus from tobacco to tobacco, although they will transmit it from tomato to tobacco and other solanaceous hosts. However, these insects will transmit the cucumber mosaic from tobacco to tobacco and from tobacco to other hosts. This virus (Nicotiana virus 1) is highly infectious by artificial sap inoculation, and it is probable that under field conditions it is transmitted chiefly by man in the various operations of cultivation.

Little is known of the nature of insect transmission of this virus. It is difficult to understand why the aphids do not transmit mechanically such a highly infectious virus. Also, the differential and specific transmission by the aphids of the various viruses that affect tobacco suggest some sort of biological relationship. There is still much to learn about the subject. The isolation of this virus in pure crystalline form (Stanley 1937a, 1938a, 1938b) should make it possible to learn a great deal more about the property of the virus and about the ways in which it may be transmitted.

Yellow Dwarf of Onions (Allium Virus 1).—Yellow dwarf is a relatively new disease of onions, having been observed for the first time in Iowa in 1927. It was very destructive in local areas and spread rapidly until methods of control were devised. The virus nature of the disease was established in 1929 by Melhus, Reddy, Henderson, and Vestal. More detailed investigations have been reported by Drake, Harris, and Tate (1932, 1933) and Henderson (1935).

The most characteristic symptoms of yellow dwarf are the yellowing, crinkling, and dwarfing of the leaves and flower stalks (Fig. 137). Underdeveloped bulbs of little commercial value are produced by diseased plants. Infected plants produce normal seed but about 30 per cent less than healthy plants.

Transmission of Yellow Dwarf.—Yellow dwarf is readily transmitted by artificial sap inoculation and by more than 50 different species of aphids. Of these the following species are most commonly found on onions and are probably of greatest impor-

tance: *Macrosiphum pisi* (Kalt.), *Aphis rumicis* L., *Aphis helianthi* Monell, *Hyalopterus atriplicis* L., and *Rhopalosiphum prunifolium* Fitch. Transmission of these insects apparently is entirely mechanical. If there is an incubation period in the insect, it is extremely short. Even though the disease is transmitted readily by artificial sap inoculation, certain insects such as grasshoppers, beetles, cutworms, coleopterous larvae, onion maggots, thrips, and bulb mites do not transmit the disease.



FIG. 137.—Yellow dwarf of onion. Four diseased plants compared with the two healthy plants in the center. (After Henderson.)

(Drake *et al.* 1933). The onion is not a preferred host for any of the aphid vectors, and none of them breeds on the onion. In addition to the aphids, the six-spotted leaf hopper (*Macrostelus divisa*) and a mealy bug (*Phenacoccus* sp.) have been reported as vectors.

The latent period of the disease in onions is about ten days. The virus is inactivated in 112 hours by aging *in vitro* and in dried onion leaves. The virus is not transmitted in the seed of the onion. It survives the winter chiefly in infected bulbs, either

volunteer plants or in commercial sets. The virus does not overwinter in the soil and is not spread to any extent by cultivation practices.

Rather effective control has been secured by a system of greenhouse indexing of onion sets and by using sets that have been grown in regions where yellow dwarf does not occur. Good results have been obtained also by spraying to control the aphids. The destruction of volunteer onions is likewise of some value. By the combined use of these practices, all based on a knowledge of the various means of transmission, the prevalence of the disease in a given area was reduced from 40 per cent in 1928 to a trace in 1934.

Cucumber Mosaic (*Cucumis Virus* 1).—Cucumbers are affected by more than one virus, but the one designated by Johnson (1927) as cucumber mosaic virus 1 is the most prevalent and is the cause of the disease described in detail by Doolittle (1920). The disease is very destructive and is a limiting factor in cucumber culture in many regions where the crop has been grown intensively over a period of years. It is destructive to greenhouse plants as well as to those grown in the field.

The virus is not confined to cucumbers but has a relatively wide host range, affecting not only other cucurbits but also many distantly related species. Furthermore many common weeds may act as carriers of the virus. It commonly survives the winter in the roots of perennial weeds, and these may serve as sources of infection for the cultivated crop. The frequency of weed infection makes practical control in the field very difficult.

The symptoms appear first on the young leaves as a typical mosaic pattern of light and darker green areas (Fig. 138). The leaves are slightly curled; and, as they grow older, their color becomes lighter yellow. Eventually they die, becoming dry, brown, and brittle. Diseased plants are greatly stunted, and the yield is strikingly decreased. Affected fruits are mottled with light- and dark-green areas which are raised above the surface to form characteristic warty protuberances. The cucumbers are distorted and undersized, the quality of the flesh is poor, and the market value is almost negligible.

Transmission of Cucumber Mosaic.—Primary infection usually arises from a virus that has survived the winter in the roots of susceptible weeds, such as milkweed (*Asclepias syriaca*), poke-

weed (*Phytolacca decandra*), and the wild ground cherry (*Physalis* sp.). The virus may be transmitted also in the seeds of the wild cucumber (*Micrompelis lobata*) and in a very small percentage of the seeds of muskmelons (Kendrick 1934).

Cucumber mosaic is readily transmitted by artificial sap inoculation, being commonly spread in pruning, picking, and cultivating operations. It is also transmitted by insects, chiefly *Aphis gossypii* Glov., although it may be transmitted less readily by *Myzus persicae* Sulz., *M. pseudosolani* Theob., *M. circumflexus* Buckt., and *Macrosiphum gei* Koch (Hoggan 1930). It is believed by some workers (Doolittle and Walker 1928) that

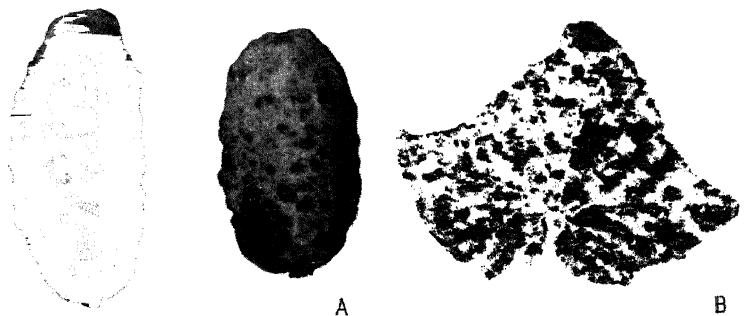


FIG. 138.—Cucumber mosaic. A, on fruits; B, on a leaf. (After Doolittle.)

aphid transmission of this virus is purely mechanical since the aphids lose their infectivity after a very short period of feeding. However, the question is open for further study because of the selective transmission of the cucumber virus by certain species of aphids from tobacco plants affected with both cucumber-mosaic virus and tobacco-mosaic virus, as demonstrated by Hoggan (1929, 1930, 1931, 1933).

In addition to aphids, the striped cucumber beetles (*Diabrotica vittata* Fabr. and *D. duodecimpunctata* Oliv.) may transmit the virus to a considerable extent. Transmission of the virus by these chewing insects apparently is purely mechanical.

Cucumber mosaic may be controlled readily in greenhouses by thorough insect control and by careful roging of infected plants as soon as the symptoms appear (Doolittle 1924). In the field, effective control is not so practical. A reasonable degree of

control has been reported in local areas by thorough eradication of the weed carriers in and about the fields (Doolittle and Walker 1926). However, this has not been practical for the individual grower.

Western Celery Mosaic (Apium Virus 1).—There are several viruses that affect celery some of which are better known for their occurrence on other economic crops. The western celery mosaic, however, is primarily a disease of celery, and its host range appears to be limited to the Umbelliferae. It is a disease of major importance to the celery industry of California. The disease has been described and studied extensively by Severin and Freitag (1938).

The symptoms of western celery mosaic are yellow, mottled foliage, twisted, cupped, and malformed leaflets, and a general stunting of the plant. A shortening of the petioles of the central leaflet and a horizontal position of the petioles of the outer leaflets gives the top of the plant a flattened appearance. Necrotic spots and streaks may develop on leaves and petioles in later stages of infection.

Transmission of Western Celery Mosaic.—The disease is readily transmitted by artificial sap inoculation, the latent period varying from 10 to 16 days. It has been demonstrated that the virus may be successfully transmitted by any of 11 different species of aphids that breed on celery in California. Transmission appears to be entirely mechanical, and there is no specific aphid vector. The virus is retained by the aphids for relatively short periods, varying from 1 to 10 hours. The aphids found on celery in California have been adequately described by Essig (1938).

There are numerous other viruses that fall within this group, most of which are transmitted in nature by several species of aphid. Owing to a lack of sufficient data on insect transmission of these viruses, it is often difficult to determine whether some of them should be placed in this or the following group.

2. TRANSMISSION BY APHIDS, SHOWING GROUP SPECIFICITY; INSECT TRANSMISSION NOT ENTIRELY MECHANICAL

Sugar-cane Mosaic (Saccharum Virus 1).—Mosaic is one of the major diseases of sugar cane wherever the crop is grown. Early symptoms consist of irregular chlorotic streaks on the

leaves, but later symptoms vary considerably (Fig. 139). The chlorosis becomes more marked, the plants are badly stunted, and necrotic lesions are often formed on the canes. The yields are greatly reduced. Sugar-cane mosaic affects corn, sorghum, millet, and several species of wild grass. In Louisiana, sorghum (*Holcus sorghum*), pearl millet (*Pennisetum glaucum*), crab grass (*Syntherisma sanguinalis*), bull grass (*Paspalum bascianum*), giant foxtail (*Chaetochloa magna*), and *Brachiaria platyphylla* are frequently found naturally infected in the vicinity of sugar-cane fields (Brandes and Klaphaak 1923).



FIG. 139.—Sugar cane leaves affected with a severe strain (A) and a mild strain (B) of mosaic. (Photographs by courtesy of I. L. Forbes.)

Transmission of Sugar-cane Mosaic.—The disease is transmitted by vegetative propagation, by artificial sap inoculation, and by insects, the most important of which is the so-called “corn aphid” (*Aphis maidis* Fitch) (Figs. 140 and 141). When it was first reported by Brandes (1920) that the virus was transmitted by this insect, the practical importance of the vector was questioned by several writers (Wolcott 1921), because sugar cane is not a preferred host of *A. maidis*. The insect cannot be reared continuously on sugar cane and feeds on the plant only when other preferred grasses are not available. Later work, however, has established the fact that this insect is an effective vector.

In nature, the aphids breed abundantly upon the wild grasses that are known to carry the virus. When these grasses are repeatedly cut down in the process of cultivation, the aphids migrate to the sugar cane and inoculate it with the virus (Brandes and Klaphaak 1923). Practical field experience has verified this sequence of events as reported in 1928 by Wolcott, who discusses the facts as they influence the control of the disease. Because cultivation of the cane cannot be dispensed with entirely, inoculation by aphids migrating from the cut grasses cannot be avoided.

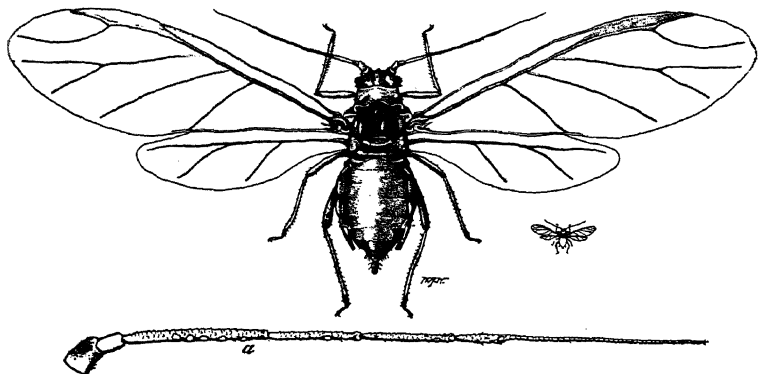


FIG. 140.—*Aphis maidis* (an adult alate male), the principal vector of sugar cane mosaic. A drawing to show its distinctive markings. (Courtesy of J. W. Ingram.)

The most successful control has been obtained through the use of mosaic-resistant selections of sugar cane.

A notable contribution to the mechanics of inoculation of plant viruses by sucking insects was made in the study of sugar-cane mosaic by Brandes (1923) who studied the histological relationships between the mouthparts of *A. maidis* and the tissues of the sugar-cane plant. He showed that the aphids invariably seek out the phloem and that a copious flow of saliva into the living tissues takes place during penetration and feeding. His conclusion that mosaic infection occurs in this way has been amply confirmed by later investigations with many viruses and vectors.

A second vector was reported by Ingram and Summers in 1936. These authors were successful in transmitting the virus

by the rusty plum aphid [*Hysteronneura setariae* (Thomas)]. This insect was not so effective as *A. maidis*, transmitting the virus only 5.2 per cent of the trials compared with 23.3 per cent transmission by *A. maidis*. The rusty plum aphid, however, was much more abundant than the corn aphid on sugar cane, this plant being one of its preferred hosts throughout the year. The corn aphid is found on sugar cane only in late winter and early spring when other hosts are scarce. The rusty plum aphid breeds also on a number of wild grasses which are common hosts of sugar-cane mosaic. For these reasons, Ingram and Summers considered it to be a vector of much significance in the spread of sugar-cane mosaic.

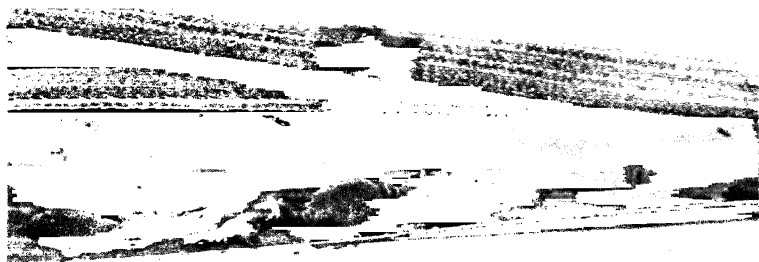


FIG. 141.—A colony of *Aphis maidis* feeding on a grass leaf. (Photograph by courtesy of J. W. Ingram.)

Ingram and Summers (1938) report the transmission of the virus in two instances with the aphid *Toxoptera graminum* Rond., which also was found on sugar cane. The number of trials was too small in this case to justify the conclusion that this insect is a vector of much significance. More recently, Tate and Vandenberg (1939), working in Puerto Rico, have incriminated another vector, namely, *Carolinaia cyperi* Ainslie, an aphid that is found abundantly in and near sugar-cane fields on its native host *Cyperus rotundus*. Since the virus is readily transmitted by artificial sap inoculation, it is probable that several other insects may be found that are capable of transmitting the disease to a limited extent.

Sugar-cane mosaic is not caused by a single virus, several strains of distinctly different virulence and causing different

symptoms have been recognized. In Louisiana, a green and a yellow strain are very prevalent, the yellow strain being much more injurious to the plant than the green strain. It has been demonstrated that a plant affected with the green strain is immune to the yellow strain (Forbes, Mills, and Edgerton, 1937). The practical importance of this discovery in sugar-cane culture has not yet been determined.



FIG. 142.—A potato plant affected with leaf roll.

Potato Leaf Roll (*Solanum Virus 14*).—Leaf roll is one of the more destructive virus diseases of the potato. It probably occurs in all countries in which potatoes are grown but appears to be more prevalent in some localities than in others. The disease is characterized by a necrosis of the phloem tissues which results in an accumulation of starch in the aboveground parts of the plant. This is accompanied by a characteristic thickening and upward rolling of the leaves, giving rise to the name "leaf roll" (Fig. 142). In pigmented varieties, the rolled leaves may show

excessive red pigmentation. Because of the interference with normal starch translocation, the tubers are small, and the yields are low.

Transmission of Leaf Roll.—The infectious nature of leaf roll was first recognized by Quanjer, Lek, and Botjes in 1916 (Quanjer 1920), and its transmission by aphids was demonstrated by Oortwijn Botjes (1920) and Schultz and Folsom (1921). Since 1921, many investigators have studied the problem of insect transmission of leaf roll, but there is yet considerable uncertainty as to the exact role of certain insects. No one has been able to transmit leaf roll by artificial sap inoculation although it is readily transmitted by grafting.

Oortwijn Botjes (1920) reported transmission by *Aphis rumicis* L. and *Myzus persicae* Sulz., and Schultz and Folsom (1921) incriminated *M. persicae* and *Macrosiphum solanifolii* Ashm. Murphy (1923) presented evidence to show that the plant bug (*Calcoris bipunctatus*) and a leaf hopper (*Typhlocyba ulmi*) were also vectors of the leaf-roll virus. Murphy and McKay (1929) verified transmission by *Macrosiphum solanifolii* and added *Myzus pseudosolani* to the list of vectors. In 1927, Elze reported that seven different species of insects were capable of transmitting the disease, five of which had not been previously incriminated. These five were *A. rhamni*, *Eupterix auratus*, *Lygus* sp., *Psylliodis affinis*, and the larvae of *Tipula paludosa*, the last-named species working below the ground level.

Smith (1929, 1931c) was unable to transmit the virus with *Calcoris bipunctatus*, *Lygus pabulinus*, *Eupterix auratus*, *Chlorita viridula*, *Psylliodis affinis*, and *Macrosiphum gei*. Smith (1934, 1937a) maintains that *Myzus persicae*, *M. pseudosolani*, and *M. circumflexus* are the chief vectors and that *M. persicae* (Fig. 143) is the most important of the three. Dykstra and Whitaker (1938) confirmed the efficient transmission of leaf roll by these three species and showed that it was transmitted less efficiently by *Macrosiphum (Illinoia) solanifolii*. The less efficient transmission by the latter species was attributed to the fact that it fed in vascular tissue in less than 50 per cent of the cases observed whereas the three *Myzus* species habitually fed in the phloem. Further careful experimentation will be necessary before these contradictory results can be explained. However, it seems that it would be necessary to assume a simple mechanical process in order

to explain the transmission of a virus by so many different kinds of insect. Inasmuch as the leaf-roll virus is not transmissible by artificial sap inoculation, mechanical transmission by all these insects would appear somewhat doubtful. At any rate, we are concerned with a virus that is not transmissible by artificial sap inoculation but is transmissible by several species of aphid. Since it has been demonstrated that there is an incubation period of the virus in *Myzus persicae* of several days, it is probable that dissemination of the leaf-roll virus in this vector is biological.

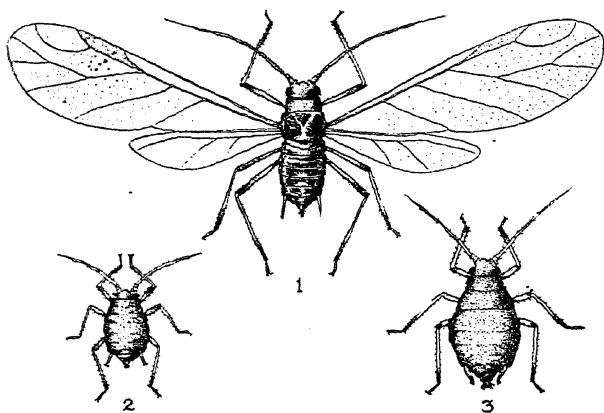


FIG. 143.—*Myzus persicae* the vector of leaf roll and more than a dozen other virus diseases. 1, adult winged female; 2, immature female; 3, adult wingless female. (After Headlee.)

The fact that the insect retains its infectivity for a relatively long period of time also lends support to this view.

The host range of leaf roll has never been studied extensively, but Dykstra (1933) has shown that the virus may be transmitted by *Myzus persicae* from potato to *Solanum villosum*, *S. dulcamara*, *Datura stramonium*, and *D. tatula* and to the cultivated tomato (*Lycopersicon esculentum*) and that these plants are important carriers of the virus in Oregon.

3. TRANSMISSION BY LEAF HOPPERS; BIOLOGICAL AND HIGHLY SPECIFIC

Curly Top of Sugar Beet and Other Plants (Beta Virus 1).—Curly top is one of the most destructive of all virus diseases of plants and probably has been studied more extensively than any

other similar disease. It is the most destructive disease of sugar beets in the regions where it occurs, often causing losses of several million dollars in a single season. It is a limiting factor in beet production in many otherwise suitable regions. Curly top is known only in the western part of North America, although a similar disease has been reported from South America (Faweett 1927). There is some question of the identity of the South American virus with that of Western North America.

Curly top has been observed since 1899, only a few years after the beet-growing industry became well established in America. It was not until the work of Ball (1906) that the disease was associated with infestations of the leaf hopper (*Eutettix tenellus* Baker). The virus nature of the disease and the role of the leaf hopper (*E. tenellus*) in transmitting the disease were thoroughly established by the work of Shaw (1910), Smith and Bonquet (1915), and Ball (1917). For many years, the disease was assumed to be limited to the sugar beet and a number of native weeds, but the known host range gradually has been extended (Carsner 1919, Carsner and Stahl 1924, McKay and Dykstra 1927, Severin and Henderson 1928, Severin 1929, 1934), until it includes many vegetable and ornamental plants as well as numerous species of wild plant. The nature and prevalence of the other hosts have been shown to have much influence on the epiphytology of curly top on sugar beets.

The symptoms of curly top vary with the host plant affected, and on sugar beets they frequently have been confused with symptoms caused by other agencies. An extensive and detailed description of the disease on sugar beets has been published by Severin (1929), who also pointed out other abnormalities that often were confused with curly top. Only a very brief description will be given here.

The earliest symptoms appear as an inward rolling of the lower and outer margins of the youngest leaves (Fig. 144). Later, the entire leaf blade curls upward along the mid-rib. Nearly always there is a distinct clearing of the smaller veins of the leaves associated with the curling. The veins are thicker than normal and more or less distorted, a condition that gives a rough warty appearance to the underside of the leaf. Affected plants are always stunted; and if infection occurs while the plant is very young, it will die prematurely.

Although the cuticle may serve as a barrier against pathogenic microorganisms, it is not always effective against the feeding and oviposition of many insects. A thick or tough cuticle, however, might easily discourage certain species of insects and cause them to show a preference for the varieties or species with a thinner

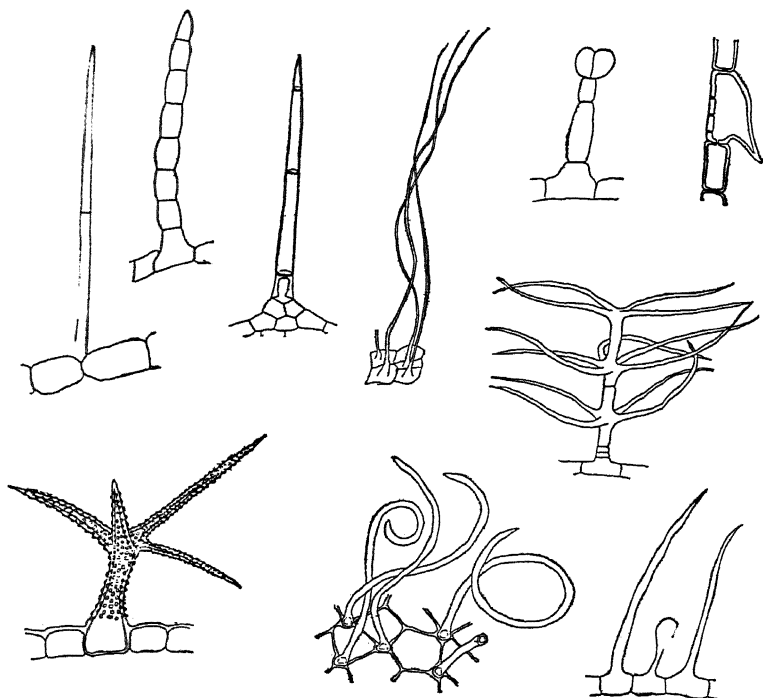


FIG. 193.—Trichomes or plant hairs from several different kinds of plants. These are all modified epidermal cells. They serve many different functions, one of which is protection against insect and fungus enemies. (After Eames and MacDaniels.)

cuticle. In such cases, if the insect were a vector of a disease, the nature of the cuticle would be an important factor in the ability of the variety to escape infection. Susceptible varieties that escape infection in this way are said to be "klendusic." Numerous examples of klendusic varieties of crop plant have been reported, but for many of them no satisfactory explanation of the

(Fig. 145) is the only known insect vector. Another species of leaf hopper (*Agallia sticticollis* Stal.) is the vector of a disease of sugar beets in Argentina which is thought by some to be identical with curly top. This insect, however, has not been reported in North America. According to Severin and Henderson (1928), *E. tenellus* does not occur in Argentina.

The transmission of the curly-top virus by *E. tenellus* is specific and biological. The beet leaf hopper is not known to transmit any other virus, and no other insect is known definitely to trans-

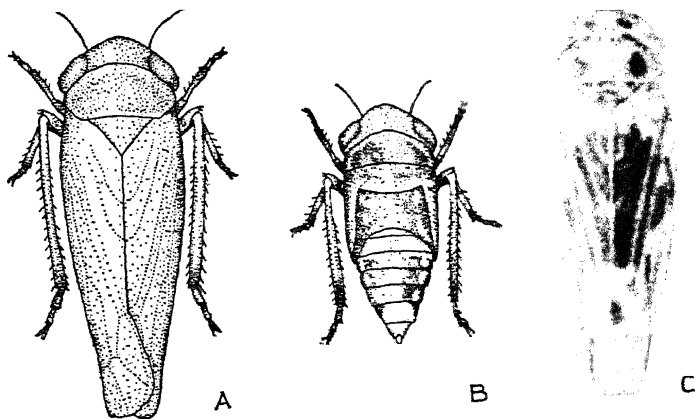


FIG. 145.—The beet leaf hopper (*Eutettix tenellus*), the vector of curly top. A, adult; B, nymph; C, adult. Approx. 25X. (A and B after Ball, C after Severin.)

mit curly top. There is a distinct incubation period of the virus in the body of the insect. The minimum incubation period first was reported as being several hours, but this has been shortened to 20 minutes when 20 to 50 viruliferous insects are used. When only 1 insect is used, the minimum incubation period is 7 hours. The latent period of the disease in the plant varies from 4 to 14 days.

The virus of curly top may survive the winter in a number of wild plants. The weeds in which the virus survives the winter are those on which the hopper feeds in late fall and early spring. The virus can survive also in overwintered sugar beets in the field from which it is transmitted to the new crop of seedlings.

The available evidence (Wallace and Murphy 1938) indicates that leaf hoppers that hibernate on infected sugar beets transmit a more virulent strain of the virus than is transmitted by those that overwinter in the deserts on wild plants.

The fate of the virus in the body of the insect vector has been studied by Freitag (1936) and by Bennett and Wallace (1938). The virus may be recovered from the blood, the salivary glands, feces, and the contents of the alimentary tract of viruliferous leaf hoppers. It is recovered most readily from the blood, a fact indicating that the blood is the chief reservoir of the virus in the insect. The virus content of the insects as well as their ability to transmit the virus decreases slowly over a period of 8 to 10 weeks when they are confined to immune plants. Viruliferous leaf hoppers, having fed for 6 hours on virus extracts, lose the power of transmitting the virus after 54 days, when transferred daily on small beet plants, but regain it after feeding for short periods on infected beets. These results indicate that the virus does not multiply in the body of the vector. It has been shown, however, by Wallace and Murphy (1938) that the virus can survive the winter in the body of leaf hoppers without any apparent change in virulence.

Swezy and Severin (1930) reported the presence of Rickettsia-like microorganisms in the epithelium and lumen of the intestinal tract of infective leaf hoppers and suggested a possible relationship with the virus. There is not sufficient evidence, however, to justify the conclusions that the microorganisms are concerned in the etiology of the disease.

The virus is not transmitted through the seed of the sugar beet. Although it invades the external tissues of the seed, it does not reach the embryo, and when the seeds germinate the seedlings do not become infected. The virus is not transmitted congenitally through the eggs of the leaf hopper, as shown by the mating experiments of Bennett and Wallace (1938).

Leaf hoppers may be made viruliferous by feeding them on extracts from infected plants. A quantity of juice is expressed from tissue and precipitated with an equal volume of 95 per cent alcohol. The precipitate contains a high percentage of the virus. It is then centrifuged, washed in 50 per cent alcohol, dried, and mixed with a 5 per cent sugar solution equal to the original volume of the juice. This in turn is centrifuged, and the super-

natant liquid is used to feed nonviruliferous leaf hoppers through a suitable membrane. This technique, perfected by Bennett (1935), has been used by Bennett (1934, 1937) and by Bennett and Esau (1936) in extensive studies of the occurrence of the virus in different tissues and its movement within the plant.

The localization of the virus in the phloem of affected plants is significant in view of the fact that the leaf hoppers feed almost exclusively upon the phloem tissues and must do so in order to

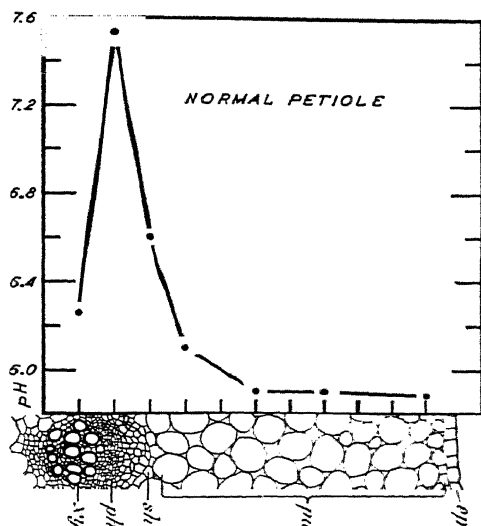


FIG. 146.—Diagram showing the pH gradient in a normal sugar-beet petiole. Note that the phloem is more alkaline than the surrounding tissues. *x*, xylem; *ph*, phloem; *sh*, bundle sheath; *pa*, parenchyma; *ep*, epidermis. This differential aids the leaf hopper in locating the phloem. (After Fife and Frampton.)

ensure infection. A plausible explanation of the means by which the leaf hoppers are able to locate the phloem has been offered by Fife and Frampton (1936). With the aid of a quinhydrone microelectrode, these workers determined the pH value of the sap of the various tissues of the beet leaf. It was found that the reaction of the phloem tissue of a normal petiole was approximately pH 7.5, that of the xylem was about pH 6.2, and that of the surrounding parenchyma tissue was slightly less than pH 6.0 (Fig. 146). There is therefore a pH gradient between the phloem

and the surrounding tissues. The fact that the saliva of the leaf hopper is alkaline, approximating that of the phloem, led to the hypothesis that the pH gradient is used by the vector in locating the phloem. This hypothesis is supported by feeding experiments in which hoppers showed preference for the alkaline solutions. Moreover, when beets are subjected to high concentrations of carbon dioxide, the pH gradient is disturbed so that

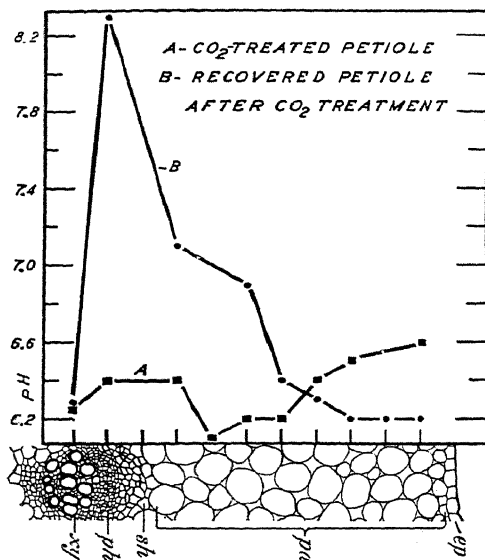


FIG. 147.—Diagram showing the pH gradient in a sugar-beet petiole treated with carbon dioxide. The differential between phloem and surrounding tissue has been lost. The leaf hoppers have difficulty in locating the phloem in treated petioles. (After Fife and Frampton.)

the hoppers are unable to locate the phloem and feed indiscriminately on any of the tissues (Fig. 147).

The leaf hopper hibernates in the adult stage chiefly as fertilized females. The males rarely overwinter. The hibernation is incomplete, there being a rest period without true dormancy. Overwintering occurs chiefly on weed hosts in the arid foothills surrounding the irrigated beet fields. They overwinter to a lesser extent in and near old beet fields. Oviposition begins about Apr. 1 on the wild hosts, the most important of which are

the probable abundance of leaf hoppers can be predicted with a fair degree of success in early spring before the beets are planted. By modification of planting practices according to the predictions, considerable losses can be avoided. Unfortunately, the predictions have not been 100 per cent correct. There are so many unknown factors (collectively termed the biological X) that the predictions are sometimes completely wrong. The prediction practice, at its best, is only an emergency measure.

A more promising outlook for eventual reduction of the losses from curly top is offered by the investigations of Piemeisel (1932), Piemeisel and Chamberlin (1936), and Piemeisel and Lawson (1937). These workers have shown that the population of leaf hoppers has been increased greatly in recent years by the increase of the weed hosts that thrive on abandoned farm lands and in overgrazed pasture land. During and shortly after the World War, when wheat prices were high, much of the marginal dry land was plowed up and sown to wheat. Following the drop in prices, much of this area was abandoned or farmed only intermittently; a great increase in weeds resulted, consisting principally in the preferred weed hosts of the beet leaf hopper. Other localities in the beet-growing regions are consistently overgrazed, a factor also favoring certain preferred weed hosts. It was shown that, when the land is not plowed or grazed, it will revert to its natural cover of plants that are not favorable hosts for the leaf hopper. Such protected land, according to these workers, will not produce economically significant numbers of leaf hoppers. They conclude that "the beet leaf hopper and curly top can be controlled provided that all lands not continuously farmed and well farmed can be restored to and maintained as good desert range. . . . Unless present practices are corrected, an increase of weedy areas, leaf-hopper populations, and curly top can be expected."

Another promising method of control of curly top of sugar beet is offered by the development of resistant varieties of sugar beets. The Division of Sugar Plant Investigations of the U.S. Department of Agriculture has introduced several new varieties of sugar beets that are fairly resistant to the disease (Owen *et al.* 1939).

With the increased use of resistant varieties and with better control of the leaf hoppers through improved range practices in sugar-beet-growing regions, the outlook for practical control of

California will infect celery whereas the one prevalent in New York will not. Severin later (1934*a, b*) discovered other differences between the two strains of the virus. Kunkel (1937) also reports the isolation of mild strains of the virus by subjecting viruliferous leaf hoppers to high temperatures. The eastern strain of the virus, according to Kunkel (1931), does not affect



FIG. 148.—Aster yellows. *A*, an aster plant affected with yellows; *B*, a healthy plant of the same age. Note the excessive axillary branching and more erect growth of the leaves and branches of the affected plant. The yellow color of the diseased plant is not shown in the picture. (After Jones and Riker.)

potatoes, but Severin and Haasis (1934) successfully inoculated potatoes with the California strain. Affected potato plants have slender purple shoots and form aerial tubers in the axils of the leaves. The incubation varied from 20 to 63 days, and the virus was not recovered from infected plants or from tubers produced by infected plants. Naturally infected plants were not observed in the field.

this destructive disease is very promising. Efforts to control the beet leaf hopper by intensive spraying practice have not been successful (Douglas, Wakeland, and Gillett 1939).

Aster Yellows (*Callistephus Viruses 1 and 1a*).—Aster yellows was first recognized and described as a destructive disease by R. E. Smith in Massachusetts in 1902. Smith was unable to find a fungus or bacterium associated with the disease and expressed the opinion that it belonged to the then little known group of virus diseases. Aster yellows along with many other diseases of ornamental plants received very little attention until many years later. In 1926, Kunkel published results of the first thorough study of the disease. The papers of Kunkel (1926, 1931, 1932, 1937) and Severin (1929, 1932, 1934*a, b*) form the chief source of our information about aster yellows and the relationship of insects to its spread and development.

Aster yellows is not limited to asters but has a very wide host range (Kunkel 1931), affecting nearly two hundred species of plants in more than thirty different families. The disease apparently is of American origin and is found throughout North America. It is rare or absent in Europe. A few reports of its occurrence in other countries have appeared in recent years (Fukushi 1930, Ogilvie 1927, Richter 1936). No accurate estimates can be given of its economic importance, but it is a limiting factor in aster culture in practically all sections of North America and because of its wide host range must be ranked as one of the more destructive diseases.

The symptoms caused by the aster-yellows virus vary considerably with the plant affected. Partial chlorosis, resulting in a yellowing of normally green tissues, is the most universal symptom. There is no mottling or mosaic pattern. A clearing of the veins of affected leaves is common. Infected plants are always stunted and often distorted, the degree depending upon how early the plant becomes infected. An excessive branching and shortening of the internodes is often observed (Fig. 148). Necrosis occurs in advanced stages, and seed produced on affected plants usually will not germinate. On asters, excessive numbers of trichomes often are formed, and a greening of the petals is not unusual. The roots show no abnormal symptoms.

There are several strains of the aster-yellows virus. Severin (1932) and Kunkel (1932) have shown that the virus prevalent in

California will infect celery whereas the one prevalent in New York will not. Severin later (1934*a, b*) discovered other differences between the two strains of the virus. Kunkel (1937) also reports the isolation of mild strains of the virus by subjecting viruliferous leaf hoppers to high temperatures. The eastern strain of the virus, according to Kunkel (1931), does not affect



FIG. 148.—Aster yellows. *A*, an aster plant affected with yellows; *B*, a healthy plant of the same age. Note the excessive axillary branching and more erect growth of the leaves and branches of the affected plant. The yellow color of the diseased plant is not shown in the picture. (After Jones and Riker.)

potatoes, but Severin and Haasis (1934) successfully inoculated potatoes with the California strain. Affected potato plants have slender purple shoots and form aerial tubers in the axils of the leaves. The incubation varied from 20 to 63 days, and the virus was not recovered from infected plants or from tubers produced by infected plants. Naturally infected plants were not observed in the field.

Transmission of Aster Yellows.—Aster yellows cannot be transmitted by artificial sap inoculation but may be transmitted by grafting. In nature, it is transmitted primarily by a single species of leaf hopper (Fig. 149). There has been some difference of opinion as to the identity of the insect. Kunkel (1926) stated that it "was identified by several authorities to which it was submitted as *Cicadula sexnotata* Fall." Dorst (1931, 1937), however, claims that *C. sexnotata* is the European species and is not found in America. The American species, according to Dorst, is *Macrostelus divisa* (Uhl.) (*C. divisa* Uhl.). Kunkel (1926) reported that specimens had been submitted to the British Natural History Museum in London and that Mr. China noted slight differences in color markings but considered the British and American insects as belonging to the same species.

Kunkel (1926), after considering evidence submitted by various entomologists, concluded that the insect is an introduced species and that "it seems probable that it was introduced into this country less than 100 years ago." If we accept this conclusion, we are faced with the problem of explaining the origin of the disease. If the disease does not occur in Europe, it probably was not introduced into North America with the insect vector. If the vector did not occur in this country 100

years ago, how was the virus disseminated before that time? Is there some still unknown vector, or is the virus of relatively recent origin? A possible explanation may be offered in the demonstration by Severin (1934b) that two other species of leaf hopper (*Thamnotettix montanus* Van D. and *T. germinatus* Van D.) may transmit the virus but not so effectively as the better known vector. However, because of the specific obligate and biologic relationship between the insect and the virus, it seems more logical to assume that the principal and perhaps the original vector is *M. divisa* and that it is native to this country.



FIG. 149.—*Macrostelus divisa*, the principal vector of aster yellows. An adult leaf hopper. (After Severin.)

The vector, like the virus it transmits, has a broad host range and is widely distributed throughout North America. It may overwinter in the egg stage, and in the milder climates it may hibernate as adult hoppers. The overwintering eggs are deposited under the epidermis of plants, such as rye, that remain green over winter. There are five nymphal instars. The eggs hatch in 11 to 13 days in summer, and the length of the nymphal period varies from 16 to 30 days. The various generations overlap to such an extent that there are no distinct broods. The insects feed chiefly upon the veins of the leaves. Both nymphs and adults are able to transmit the virus, but an incubation period of at least 10 days in the insect is required before it becomes infective. Therefore, the disease is rarely transmitted by the early nymphal instars. Some individual insects remain viruliferous for their entire life, but others may retain the virus for a short time only. Kunkel (1937) recently has shown that viruliferous insects held at high temperatures (31 degrees centigrade or above) for several days lose the virus and become non-infective. He thinks that the vectors may commonly lose the virus in the hot weather of midsummer, thus accounting for the observed slower spread at that season of the year. Viruliferous individuals appear normal in structure (Dobrosky 1929, 1931), and the virus has not been detected in the body of the insect by histological methods. The virus is not congenitally transmitted through the egg.

The latent period of the virus in the plant averages 18 days at normal temperatures. The virus overwinters in susceptible perennial plants such as *Chrysanthemum*, *Sonchus*, *Asclepias*, *Erigeron*, and *Plantago*. It is not transmitted through the seed of the aster. Seeds produced on affected plants usually do not germinate.

Aster yellows can be controlled only by protecting the plants from the hoppers. As *M. divisa* is not a natural greenhouse insect, the control of the disease in greenhouses is not difficult. Spraying has not proved effective in protecting the plants from the insect. It is the common practice where the disease is prevalent to grow asters under large cages made of cloth netting or screen wire. This method gives very satisfactory control (Jones and Riker 1931) and is used commercially on an extensive scale.

Streak of Corn (Maize) (*Zea Virus 2*).—Corn (*Zea mays* L.) in Africa is affected with a destructive virus disease known as "streak." The disease was first described briefly in 1901 by Fuller. It was recognized as a virus in 1925 by Storey, who has studied it extensively since that date. It has not been reported from any locality outside Africa, where it is one of the major diseases of corn. No accurate figures are available as to the amount of injury. Storey has reported as high as 100 per cent infection in certain fields; but much of the disease appears late in the season, and late infections are less injurious than early ones.

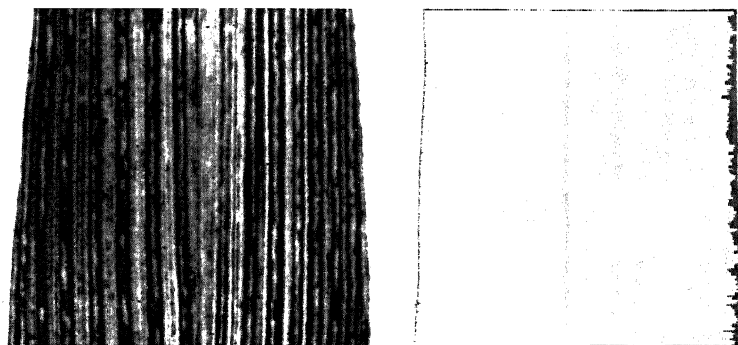


FIG. 150.—The streak disease of corn. A portion of a leaf affected with the disease (left), compared with a similar portion of a healthy leaf. (After Storey.)

The symptoms of streak appear first as very small colorless spots scattered over the leaves. These gradually elongate into streaks of yellow chlorotic tissue along the veins (Fig. 150). Since the virus is not seed-transmitted, the first leaves often escape infection until they are fully differentiated and consequently no streaks develop. The host range of streak is not fully known, but in addition to corn a number of wild grasses are also affected.

The virus is incapable of causing permanent infections on sugar cane, which appears to be highly resistant. There is a similar streak on Uba cane, but the latter disease causes only mild symptoms on maize. Storey considers the two viruses to be different. Still another virus disease of maize has been reported by Storey (1937). This causes a mild mottling of

affected leaves and has been named "mottle." Although the mottle virus is transmitted by the same insect vectors that transmit streak, Storey considers it to be unrelated to the streak virus.

Transmission of Streak.—The methods of transmission of this virus have been investigated very thoroughly by Storey (1925, 1926, 1928, 1932a, 1933). The virus is not transmitted through the seed of the corn plant, and all efforts to transmit it by artificial sap inoculation have failed. It is transmitted with a high degree of efficiency by the leaf hopper [*Cicadulina mbila* (China)] and Storey (1937) has recently reported that two closely related species (*C. zeae* China and *C. storeyi* China) are also able to transmit it. *Aphis maidis*, the principal vector of sugar-cane mosaic, *Peregrinus maidis*, the vector of a mosaic of corn in Hawaii, and about twenty additional species of insects found on corn are unable to transmit the streak virus. The transmission appears to be biological and specific for the leaf hoppers of the genus *Cicadulina*.

There is a minimum incubation period of the virus in the vector, varying from 6 to 12 hours at 30 degrees to 84 hours at 16 degrees centigrade. The latent period in the corn plant varies from $3\frac{1}{2}$ to $7\frac{1}{2}$ days in rapidly growing young plants. It is somewhat longer in older plants, depending on the temperature and the part of the plant inoculated.

The leaf hopper may acquire and transmit the virus as either nymph or adult but does so more readily as a nymph. After the virus has been acquired by the nymph, the insect retains it for the rest of its life. The virus can be acquired by feeding for 1 hour on an infected plant, but greater success results from somewhat longer feeding periods. One insect is sufficient to transmit the virus, but a higher percentage of infection is often obtained when several insects are used.

Infection follows feeding by viruliferous leaf hoppers on either young or old tissues. Symptoms appear sooner if young leaves are inoculated and may not appear at all on the older parts although the younger tissues of the same plant will show them. The virus travels down the leaves from the point of inoculation and up the stem until the growing point is reached. The rate of travel varies considerably, but it may be as fast as 40 centimeters per hour. The virus is localized in certain tissues of

infected plants. When the leaf hoppers are fed on the normal green areas of the leaf, they do not acquire the virus. It can be acquired only by feeding on the yellow tissues.

The virus is filterable through Chamberland L1 filters, Berkfeld *r* and *n* filters, and to a slight extent through Chamberland L3 filters but will not pass a Seitz E. K. filter disk. When insects are fed through membranes on the filtered juice, they become viruliferous.

Certain individual leaf hoppers appear never to become viruliferous even though they may feed indefinitely on infected plants. This phenomenon has been investigated by Storey (1932*a*, 1933), who has shown that the ability or inability to become viruliferous is an inherited character, being inherited as a dominant sex-linked character. An individual insect that is unable to transmit the virus is said to be "inactive"; one that is capable of transmitting the virus is "active." When an active female is crossed with an inactive male the F_1 progeny is entirely active. The F_2 progeny of this cross consists of active females and both active and inactive males. When inactive females are crossed with active males, the F_1 progeny consists of active females and inactive males. Both active and inactive males and females appeared in the F_2 progeny of this cross. These results support the hypothesis that the male insect is heterozygous for sex and that the factor for activity is dominant to that of inactivity and linked with sex.

Storey (1933) made an exhaustive study of the mechanism of transmission of the virus by *C. mbila*, and his results throw considerable light on the question. A technique was devised by which insects could be made viruliferous by injecting the virus obtained from viruliferous insects into the body cavity of other insects. With the aid of this technique, a study was made of the distribution of virus in the body of the vector. The virus was detected in the contents of the rectum of viruliferous or active insects provided that the insect had recently fed upon a diseased plant. It was not found in naturally voided feces. The virus was always present in the blood of viruliferous individuals, and it could be detected in the blood before the normal incubation period had elapsed. The virus could be demonstrated in the rectum of inactive insects that had recently fed on diseased plants but was never found in the blood of such insects. Inactive

(1938) added the varieties Indian Summer, Pyne Imperial, and Pyne Royal to those on which *A. rubi* could not maintain its population. Reproduction was slow and the population remained small on Antwerp, Herbert, Marcy, and Newburgh.

The nature of the resistance to the aphids was not determined, although it was stated that "the behavior of *Amphorophora rubi* when confined to Lloyd George plants under cages indicates that resistance probably results from a lack of suitable food for the insect rather than the presence of an actively repellent substance." When the variety Lloyd George was crossed with susceptible varieties, the aphid-resistant character was inherited. The significance of a knowledge of these relationships in the breeding of improved varieties of raspberries is obvious. A true mosaic-resistant variety would be desirable; but if this is not easily obtained, a klendusic variety would be of great economic value. Even though only partial control could be obtained by the use of such varieties, the efficiency of control of mosaic through certification would be greatly increased by the slower rate of spread of the disease on klendusic varieties.

A similar relationship has been shown to exist among varieties of cranberry and the leaf hopper (*Euscelis striatulus*), vector of cranberry false blossom (Wilcox and Beckwith 1933). The degree of susceptibility of cranberry varieties under field conditions and the relative rate of spread of the disease were shown to be directly correlated with their attractiveness to the leaf hopper.

One of the best known examples of resistance to insect injury in plants is that of the native American grapes to the grape *Phylloxera* (*Phylloxera vastatrix* Planchon). This insect is a native of Eastern North America and commonly infests the various species of wild grape that, through natural selection, have acquired a high degree of tolerance to the insect. When the *Phylloxera* was introduced into France about 1860, the European species of grape proved to be so susceptible that within 25 years nearly one-third of the vineyards of France had been destroyed by it. The *Phylloxera* attacks both leaves and roots, but the greatest injury is caused by those which feed upon the roots. Numerous soft, watery galls are formed on the infested roots. These galls soon decay and greatly impair the root system, and the death of the entire plant is often the result.

Fukushi (1937) who has shown that the leaf hopper (*Deltocephalis dorsalis* Motsch.) also is a vector of the disease has pointed out that Takata, as early as 1895, claimed that this insect was responsible for the disease.



FIG. 151.—Rice dwarf. A, a healthy plant and a diseased plant growing in the same pot; B, a single rice leaf showing the interrupted chlorotic streaks that are characteristic of affected plants. (After Fukushi.)

Dwarf of rice has been destructive in Japan for many years. Katsura reports historical records of famines caused by rice failures as early as 1773 and attributes the failure to the dwarf disease. More recent crop failures since 1883, involving famine and many deaths, can be attributed definitely to dwarf. The disease has not been reported from any other country.

The symptoms have been described in detail by Fukushi (1934a) who has done the best of the more recent work on the

disease. As the name indicates, there is a marked stunting of affected plants, accompanied by a correspondingly reduced yield of grain (Fig. 151). The first symptoms usually appear in late June after the plants have been transplanted into the fields and when they are 6 to 8 inches high. Small chlorotic spots appear along the veins of the new leaves. The spots gradually elongate, forming continuous or interrupted streaks parallel with the veins. Root development is arrested, and few or no fruiting panicles are formed, depending upon how early the plant is infected.

In addition to rice, the disease affects the following plants (Fukushi 1934b): *Panicum milaceum* L., *Echinochloa crus-galli* Beauv. subsp. *colona* var. *edulis* Honda., *Poa pratensis* L., and *Alopecurus fulvus* L. Rye, wheat, and oats are slightly susceptible; but corn, Italian millet, barley, and sorghum are not infected.

Transmission of the Dwarf Disease of Rice.—The leaf hoppers (*Nephotettix apicalis* Motsch. var. *cincticeps* Uhl. and *Deltocephalus dorsalis* Motsch.) are the only known vectors of the virus. The disease is not transmissible by mechanical sap inoculation or through the seed of rice. There is no evidence of transmission through the soil.

Our knowledge of the transmission of the virus of rice dwarf is based almost entirely on work done with *N. apicalis*, the other vector having been studied very little. Transmission by the leaf hoppers apparently is biological. An incubation period in the insect body is necessary although the minimum time is variable and has not been definitely determined. A minimum feeding period of 3 days is necessary for a nonviruliferous leaf hopper to pick up the virus. Some individuals do not become viruliferous even after prolonged feeding on infected plants.

The vector (*N. apicalis*) (Fig. 152) is of particular interest because in it the virus is transmitted from one generation to the next congenitally through the egg. Fukushi (1933, 1939) has shown by crossing experiments that transmission through the egg occurs only when the female parent is viruliferous. In other words, when noninfective females are crossed with infective males the virus is not transmitted to the progeny; but, on the other hand, when infective females are crossed with noninfective males a relatively large percentage of the progeny are infective (see Table II). Fukushi believes that the eggs become infected in the early stages of their development in the ovary, but appar-

ently some of the ova may escape infection. This is the only known example of the congenital transmission of a plant virus. Transmission of the virus through the egg was proved by picking up the nymphs bred from infective female leaf hoppers as soon as they hatch and before they have had time to feed on the infected plant and transferring them to healthy plants. Congenital transmission was demonstrated for seven successive generations in experiments reported by Fukushi in 1939.

There are four to five broods of the leaf hopper (*N. apicalis*). It usually overwinters as nymphs feeding on *Astragalus sinicus* L.

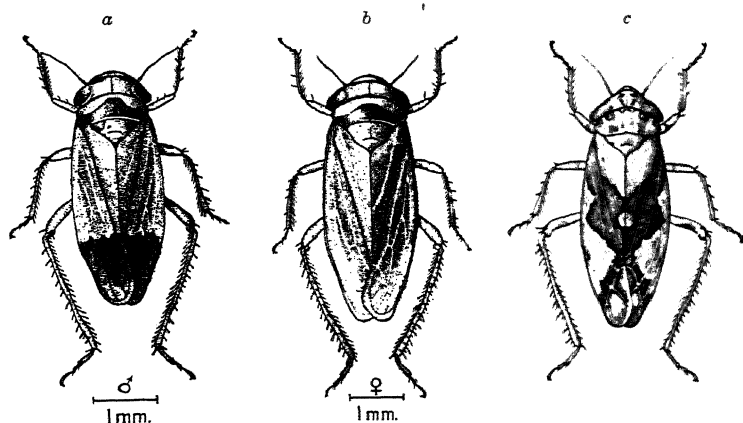


FIG. 152.—The vectors of rice dwarf. *a* and *b*, *Nephrotettix apicalis* var. *cincticeps* Uhl.; *c*, *Deltocephalis dorsalis* Motsch. (Illustrations by courtesy of T. Fukushi.)

and wild grasses, and matures in April or May. Twenty-seven to thirty-three days are required for the completion of a life cycle. The virus may live over winter in viruliferous nymphs or in susceptible perennial hosts. *Alopecurus fulvus* and *Astragalus sinicus* L. are potential overwintering host plants, but it has not been proved definitely that the virus survives in either of them. Fukushi (1934a) states that effective control of the dwarf disease is being accomplished by controlling the leaf hoppers.

Peach Yellows (Prunus Virus 1).—Peach yellows has been recognized for more than a hundred years as one of the most destructive diseases of peaches. The disease is found only in North America and appears to be limited to the eastern part of

the continent, extending westward through the upper Mississippi Valley and Great Lakes region to the Mississippi River. The host range of the virus is not fully known, but Smith (1888) states that it affects "peaches, nectarines, almonds, and apricots." Manns (1933, 1934, 1935, 1936) and Manns and Manns (1935) have shown that the wild plums (*Prunus myrobalan* and

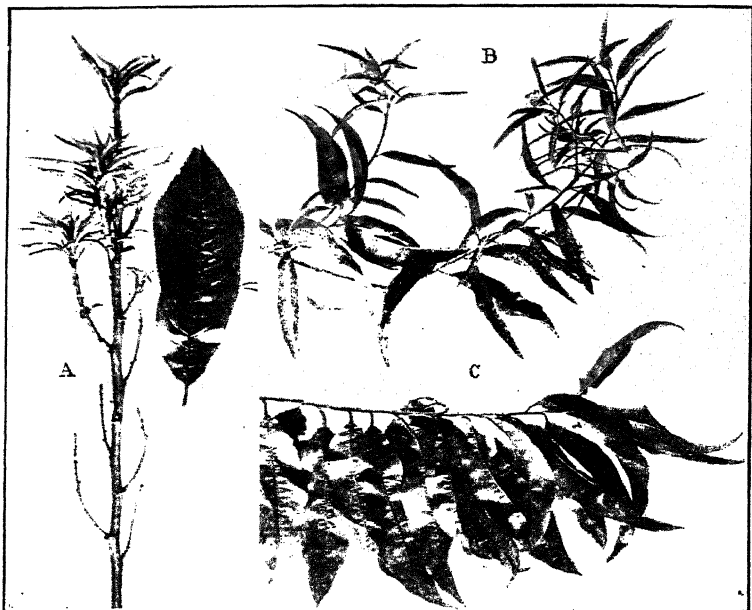


FIG. 153.—Peach yellows. A, a typical infected shoot showing the upright habit and small narrow yellow leaves with a normal leaf for comparison; B, a terminal shoot infected with yellows; C, a healthy terminal. (After McCubbin, *Pennsylvania Dept. Agr. Bul.* 382.)

P. munsoniana), the wild peach (*P. persicae*), and the Japanese plum (*P. salicina*) are carriers of the virus.

The disease is characterized by small pale yellow leaves borne on slender excessively branched twigs that tend to grow more erect than normal, forming a so-called "witches'-broom." (Fig. 153). The leaves may droop and curl upward and inward along the margins. The fruit on affected trees ripens prematurely and is often speckled or blotched with red. The texture of the

fruit is watery, and the flavor is insipid. An infected tree never recovers and may serve as a center of infection for further spread.

Transmission of Peach Yellows.—Smith, in 1888, was the first to demonstrate the infectious nature of the disease. He proved that it was transmitted by grafting or budding but not by direct sap inoculation. He was unable to discover the method of spread



FIG. 154.—*Macropsis trimaculata*, the vector of peach yellows. (After Kunkel by courtesy of the Boyce Thompson Inst. Plant Research.)

in nature, and this aspect of the problem has defied the efforts of many capable investigators since 1888.

In 1933, Kunkel demonstrated that the vector of peach yellows is the plum leaf hopper [*Macropsis trimaculata* (Fitch)] (Fig. 154). Unsuccessful attempts to transmit the virus were made with the following species of insects: *Myzus persicae* (Sulz.), *Aphis persicae-niger* Smith, *Lygus pratensis* (L.), *Conapsis*

citrosa Say, *Pseudococcus citri* Risso, *P. longispinus* (Targioni), *Philaenus leuothalmus* (L.) var. *pallidus* (Zett.), *Thelia trimaculata* (Fabr.), *Graphocephala coccinea* (Forst), *Empoa rosae* (L.), *Jassus olitorius* Say, *Fieberiolla flori* (Stal.), and *Erythroneura obliqua* Say. Manns (1935, 1936) has failed to transmit the virus with *Ceresa bulbatus* Fabr., *Gypona octolineata* var. *striata* Burm., *Thelia uhleri* Stal., *Phlepsius irroratus* Say, *Ascanolonia conica* Say, *Ormensis pruinosa* Say, and *Cicada septendecim* L. Hartzell (1935) tested 48 different species of insects and was unable to transmit the virus with any except *M. trimaculata*. Inasmuch as the insects tested include the most common sucking insects found on the peach in the region where yellow occurs, it is likely that *M. trimaculata* is a specific and obligate vector of the disease.

Peach yellows is not transmissible by artificial sap inoculation, according to the work of Blake, Cook, and Connors (1921), Manns (1924), Hartzell (1935), and several other investigators. Although occasional claims of seed transmission have been made, the evidence clearly indicates that it is not transmitted either through the seed or through pollen.

The discovery of the vector of peach yellows may prove very significant in the practical control of the disease. As pointed out by Hartzell (1935), *M. trimaculata* produces only one generation a year, and any individual leaf hopper killed would not be replaced during the current season. The population trend of the insect on wild plum and peach in New York in 1934 is given in Fig. 155. The nymphs are not so active as most species of jassids and should be relatively easy to kill by contact sprays. Manns (1934, 1935) has demonstrated that *M. trimaculata* transmits the virus of "little peach" as well as that of peach yellows.

The percentage of success in transmitting the virus of peach yellows with *M. trimaculata* has always been relatively low, a fact indicating that all individuals may not be capable of transmitting it. The length of the incubation period in the insect has not been definitely determined, but it may vary from 8 to 26 days (Hartzell 1936). Both nymphs and adults are able to acquire and transmit the virus. Intracellular inclusions have been demonstrated in the intestinal walls and in the salivary glands of viruliferous leaf hoppers by Hartzell (1937).

The plum, including wild species, is a favorite host of *M. trimaculata*, and being susceptible to yellows it constitutes an important source of infection for peach orchards. The destruction of these breeding places may be of value in the control of the disease. Manns (1934) reported that the Japanese varieties (*Prunus salicina*) are hosts of the vector and that the variety Abundance is a symptomless carrier of peach yellows and little peach. This variety is considered especially dangerous as a source of infection to peaches. Manns has expressed the opinion

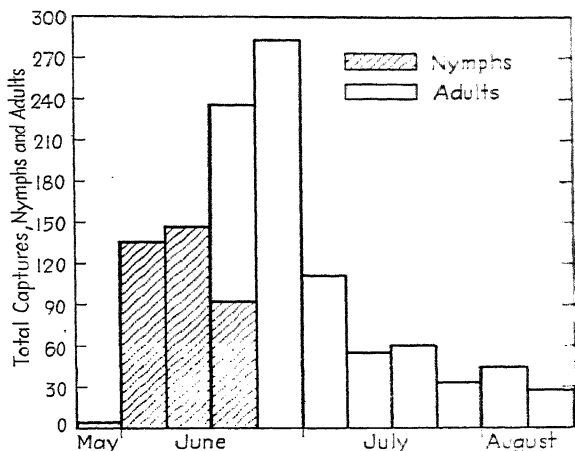


FIG. 155.—The seasonal abundance of *Macropsis trimaculata*, the vector of peach yellows, at Yonkers, N. Y., in 1934. Note that there is only one brood, which reaches its greatest numerical abundance during the last week of June. (After Hartzell.)

that the virus of peach yellows may have been introduced into America on the oriental plums.

False Blossom of Cranberry (*Vaccinium Virus 1*).—This destructive virus disease of cranberries was first observed in Wisconsin but is now present wherever cranberries are grown in the United States. It is not known outside North America. The disease, as the name indicates, causes a malformation of the blossoms that usually renders them sterile (Fig. 156). The normal floral elements are often replaced by leaflike structures that manifest a variety of abnormal modifications. Axillary buds are stimulated to growth resulting in a witches'-broom effect.

The yield of fruit on infected plants is greatly decreased, and fruit that does mature is of inferior quality. A good description of the disease has been published by Dobroscky (1931) who also demonstrated its method of transmission.

Transmission of False Blossom.—The virus of false blossom has not been transmitted by artificial sap inoculation. It also has not been transmitted by grafting because of the inability to obtain satisfactory graft unions. In nature, it is transmitted



FIG. 156.—False blossom of cranberry. A, a healthy shoot with normal blossoms; B, a diseased shoot with sterile blossoms. (After Beckwith and Hutton.)

by the blunt-nosed leafhopper (*Euscelis striatulus* Fall.) (Fig. 157) as demonstrated by Dobroscky (1929, 1931). No other vector is known.

The spread of false blossom from Wisconsin through the regions of cranberry production has been rapid. The virus was introduced into new regions on infected plants, and further spread has been effected by the leafhopper vector. An enlightening account of the spread of the disease and the part played by the vector *E. striatulus* has been given by Stevens (1930). The spread of the disease was very rapid in the decade between 1920 and 1930,

a fact attributed in part to the use of more susceptible varieties and in part to an increase in the abundance of the insect vector. The increased abundance of the insects was attributed to certain changes in cultural practices that resulted in less effective control of the insects. For example, during the period of greatest spread of the disease there was a decrease in the practice of insect control through late flooding of the bogs.

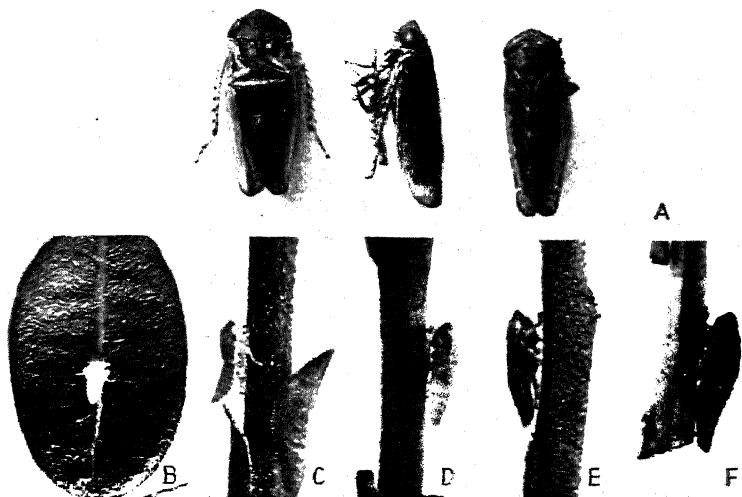


FIG. 157.—The blunt-nosed leaf hopper, the vector of cranberry false blossom. A, three adults, showing ventral, lateral, and dorsal views; B to F, the five successive nymphal instars. (After Beckwith and Hutton.)

Although false blossom is present in the bogs of the Pacific Coast states where it was introduced on diseased plants, it has spread very little and is of minor importance. The failure of the disease to spread in these regions can be explained by the absence of the vector, which has not been found in Western United States.

Euscelis striatulus is the most abundant leaf hopper found in cranberry bogs. It occurs in small numbers on wild cranberries and related plants, but is most abundant in cultivated bogs. There is only one brood each year. The eggs are deposited under the bark of the young shoots in August and September, remaining

dormant over winter and hatching in late May or June. There are five nymphal instars, each lasting about seven days (Fig. 157). The adults, therefore, are most numerous in July and August. The life history of the insect in relation to the false-blossom disease has been studied by Beckwith and Hutton (1929).

Control of false blossom is being accomplished largely through the growth of resistant varieties such as McFarlin and through better control of the insect vector. The most practical method of doing this is by flooding the bogs in late June just after the eggs have hatched; but since some injurious effects have followed

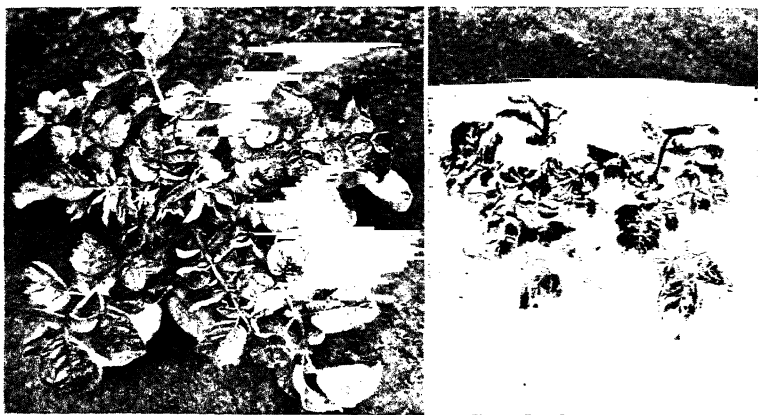


FIG. 158.—Potato yellow dwarf. Two plants showing symptoms of different degrees of severity. (Photographs by courtesy of the Wisconsin Agr. Exp. Sta.)

flooding at certain times, further studies in control are desirable. Some success has been obtained by spraying in late June with pyrethrum sprays (Beckwith and Hutton 1929).

Potato Yellow Dwarf (*Solanum Virus 16*).—Yellow dwarf was first recognized in 1917 in New York and was described by Barrus and Chupp in 1922. The disease is now known to be present to some extent throughout the northeastern part of the United States. It has not been reported from outside continental North America. It is not uniformly distributed but appears to be very destructive in certain local regions and absent in others.

The symptoms of yellow dwarf vary extensively (Fig. 158). Black (1937) recognized two distinct types of symptom, referred

to as acute and chronic. The acute symptoms are found on newly inoculated plants and consist of extreme dwarfing caused by the death of the apical meristem, accompanied by pronounced yellowing of the leaves. There is an apical internal necrosis of the stem and a necrotic spotting of the internal tissues of the tubers. The chronic symptoms appear later after a state of equilibrium between virus and susceptible has been reached. They consist of a spindly growth with slightly mottled and ruffled leaves. The necrosis of apical meristem and the extreme dwarfing are absent. The virus is not attenuated in the chronic stage, as proved by the acute symptoms that develop when healthy plants are inoculated by grafting with plants showing the chronic symptoms.

The symptoms are strikingly modified by temperature, as shown by Goss and Peltier (1925), Black (1937), and Walker and Larson (1938 and 1939). The symptoms are most acute at the higher temperatures (24 to 28 degrees centigrade). At soil temperatures of 16 degrees centigrade, the symptoms are almost entirely masked.

Transmission of Yellow Dwarf.—This disease is transmitted by grafting and by insects, but it has not been transmitted by direct sap inoculation. The virus is perpetuated through the tubers of infected plants. There have been some differences of opinion as to the insect vectors of yellow dwarf. Koch (1934) reported transmission by *Myzus persicae* Sulz., and Muncie (1935) claimed that *Macrosiphum solanifolii* Ashm. and *Empoasca fabae* Harris are vectors of yellow dwarf. Black (1937), however, has been unable to confirm the work of Koch and Muncie in carefully controlled experiments. Black has presented convincing data that the virus is readily transmitted by the clover leaf hoppers (*Aceratagallia sanguinolenta* Prov.) (Fig. 159). He was unable to transmit it with *M. persicae*, *E. fabae*, *Macro-*



FIG. 159.—The clover leafhopper (*Aceratagallia sanguinolenta*), the vector of potato yellow dwarf. $\times 52$. (After Black.)

siphum solanifolii, *Phenacoccus gossypii* Towns. and Ckl., *Pseudococcus citri* Risso, *Deltocephalus mimicus* Say, *Lygus pratensis* L., and potato flea beetles. The disease has not been found occurring naturally outside the known range of the clover leaf hopper which includes practically all the United States east of the Rocky Mountains. Transmission by the clover leaf hopper has been confirmed by Walker and Larson (1939).

The complete host range of the virus is not known, but the important leguminous hosts of the leaf hoppers are susceptible. Black has shown that *Trifolium pratense* L., *T. hybridum* L., *T. repens* L., and *T. agarium* L. are susceptible to the yellow dwarf virus.

The clover leaf hopper lives over winter in the adult stage and is to be found in clover and potato fields throughout the growing season. Black has shown that the virus is preserved over winter in the body of the vector and that the overwintered vectors may be actively viruliferous. The proximity of clover fields to potato fields in New York has much influence on the epiphytology of the disease on potatoes. Visual evidence of the spread of the virus from clover fields is not uncommon. The virus is not transmitted through the seeds of clover. For this reason the amount of the virus in clover fields increases with the age of the field. Two- and three-year-old fields are more dangerous as infection sources than one-year fields. This relationship between clover fields and yellow dwarf in potatoes was not observed in Wisconsin by Walker and Larson (1939).

The Fiji Disease of Sugar Cane (*Saccharum* 2).—The Fiji disease has been known for more than thirty years. It was first observed in the British Crown Colony of Fiji from which it has derived its name, and it was first described adequately by Lyon in 1910. It is now known to occur in Australia, the Philippine Islands, Java, and New Guinea where it is one of the major diseases affecting sugar cane.

The most characteristic diagnostic symptoms of the Fiji disease are the small elongate galls formed on the veins on the under surface of the leaves. The galls are smooth and lighter green than the normal tissue (Fig. 160). The leaves are distorted, and the entire plant is stunted. It spreads rapidly and often may result in almost 100 per cent loss in a susceptible variety.

Transmission of the Fiji Disease.—The disease is transmitted vegetatively through sets and ratoons from diseased canes, and recovery has never been observed. It has never been transmitted by artificial sap inoculation. In the field it is transmitted by leaf hoppers of the genus *Perkinsiella*. Proof of its transmission by these insects was obtained independently and at approximately the same time by Ocfemia (1933, 1934) in the Philippines and by Mungomery and Bell (1933) in Queensland, Australia.

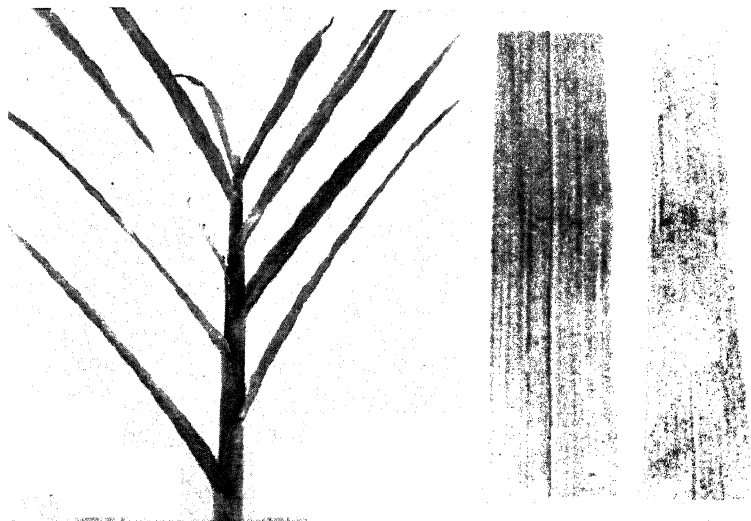


FIG. 160.—The Fiji disease of sugar cane. A, a single stalk showing the stunted growth and shortened internodes; B, the long, light-colored elevated leaf galls that are characteristic of the disease. (Photographs by courtesy of G. O. Ocfemia.)

In the Philippines the disease is transmitted by *P. vastatrix* Breddin and in Queensland by *P. saccharicida* Kirk. In the latter case, the disease was transmitted only by the nymphs while in the Philippines, according to Ocfemia, it was transmitted by adult insects. In a later paper, Ocfemia and Celino (1939) reported that the second, third, fourth, and fifth instar nymphs of *P. vastatrix* could transmit the virus but the first instar nymphs could not do so. The highest percentage of successful transmission was obtained with the fifth instar nymphs and the incu-

bation period was shorter. These authors also presented evidence to show that the virus is not congenitally transmitted through the eggs of *P. vastatrix*.

The bionomics of the sugar-cane leaf hopper (*P. vastatrix*) (Fig. 161) has been studied extensively by Urbino (1927). Sugar cane is the only known host of the insect in the Philippines. It is considered a major pest of this crop. The insect is known only in the oriental region of the Eastern Hemisphere. Since sugar cane is an introduced species in the Philippine Islands, Urbino believes that some native grass served as the primitive host of the insect.



FIG. 161.—*Perkinsiella saccharicida*, one of the two known vectors of the Fiji disease of sugar cane. (Photograph by courtesy of G. O. Ocfemia.)

4. TRANSMISSION BY THRIPS; BIOLOGICAL AND SPECIFIC

Spotted Wilt (*Lycopersicum* Virus 3).—Spotted wilt, a destructive disease of tomatoes, was first described in Australia in 1919 by Brittlebank. It is considered the most destructive disease of tomatoes in that country and lately has been reported from New Zealand (Chamberlain and Taylor 1936), England (Smith 1932), Canada (Berkeley 1935), and from several localities in the United States (Doolittle and Sumner 1934, Gardner and Whipple 1934). The disease is not confined to the tomato but has a very wide host range, including many weeds as well as agricultural crops. It is said to be very prevalent on many species of ornamental flowering plants (Bald and Samuel 1931; Gardner, Tompkins, and Whipple 1935; Snyder and Thomas

1936). In respect to host range, the virus is similar to that of aster yellows and curly top, but it differs strikingly in other respects.

The term "spotted wilt" is, in some respects, a misnomer, as pointed out by Samuel, Bald, and Pittman (1930); for affected plants are not always spotted, and they never wilt as a result of



FIG. 162.—A tomato leaf affected with spotted wilt. The lower leaflets show the typical bronze mottled area. (After Samuel, Bald, and Pittman.)

the disease. The most characteristic symptom on tomatoes is the sudden appearance of bronze-colored areas on the younger leaves of rapidly growing plants (Fig. 162), followed by an equally sudden cessation of growth. The detailed symptoms vary greatly according to the species of plant affected, the variety, its age, and the prevailing temperature. Plants infected while very young may die prematurely, but older plants live for

months in a dwarfed condition. In warm weather, excessive lateral branching may occur, giving the plant a bunchy appearance. Little or no fruit is formed on severely affected plants. Fruit, when produced, ripens slowly and is of poor quality. Often, distinct lesions occur on the fruit in the form of concentric circles of discolored tissue (Fig. 163). These concentric rings are the typical symptoms produced by the virus on many other



FIG. 163.—A tomato fruit affected with spotted wilt. (After Samuel, Bald, and Pittman.)

species of plants where the disease has been called "ring spot" (Smith 1932).

Transmission of Spotted Wilt.—Spotted wilt is readily transmitted by artificial sap inoculation, but in nature it is transmitted chiefly by several species of thrips (*Thrips tabaci* Lindeman, *Frankliniella lycopersici*,¹ *F. occidentalis* Perg., and *F. moultoni* Hood). Spotted wilt is the first virus disease shown to be transmitted by thrips. Pittman in 1927 presented evidence to show

¹ There has been some question as to the correct identification of this insect. It was first identified as *F. insularis* Franklin, but Steele (1935) concluded that it was a new but unnamed species. It was finally identified as *F. lycopersici* n. sp. by Andrewarther (1937).

that the disease is transmitted by *T. tabaci*, and in 1930 Samuel, Bald, and Pittman showed that *F. lycopersici* is an effective vector. These authors concluded that *T. tabaci* was not a true vector and that perhaps Pittman's identification had been erroneous. Smith (1932), however, proved that *T. tabaci* is an effective vector in England, and now both insects are recognized as vectors in Australia (Bald and Samuel 1931). *Frankliniella occidentalis* and *F. moultoni* have been reported as vectors of the disease in California (Bailey 1935, Essig and Michelbacher 1936). Many other insects have been tested, but the thrips are the only vectors known.

Even though the disease may be transmitted by artificial sap inoculation, transmission by thrips is not merely mechanical. A minimum incubation period of 5 to 7 days in the insect is required. The virus may be transmitted by both larvae and adults although adults cannot become infective by feeding on diseased plants. The virus can be acquired from the plant only by the larval thrips; but it survives through metamorphosis, and the adults may become infectious without having fed as adults on a diseased plant. The cause of this peculiar relationship is not known. The life of the adult is considerably longer than the maximum incubation period of the virus in the insect's body, and so it cannot be explained on the basis of a long incubation period. Inasmuch as the adults may transmit the virus acquired in the larvae stage, the size of the mouth parts constitutes no limiting factor in the movement of the virus particles. The explanation of the inability of adult thrips to acquire the virus by feeding must await further knowledge of the physiology of the insect.

After an individual insect has acquired the virus, it retains it for several weeks, although its transmission during this period is often erratic. The virus is not transmitted congenitally through the eggs of thrips or the seeds of tomato. The latent period of the virus in the tomato is 8 to 18 days, and its length is influenced by temperature and other factors, the average being approximately 12 days. The virus, in extracted juices, is short-lived, losing its potency in 3 hours. It is also sensitive to heat, being inactivated in 10 minutes at 42 degrees centigrade.

Thrips are very small insects of the order Thysanoptera. The adults are dark brown or black. They live on a wide range of

host plants and are often destructive independently of virus transmission. They feed on the epidermis of leaves and other succulent tissues with a rasping and sucking action. Feeding wounds usually stand out as light-colored irregular spots on the leaf surface.

Frankliniella lycopersici (Fig. 164) reproduces both sexually and parthenogenetically. The minute colorless eggs are deposited beneath the epidermis near a vascular bundle. The eggs hatch in about twelve days. The larvae are light-colored and almost translucent. They feed on leaves immediately after

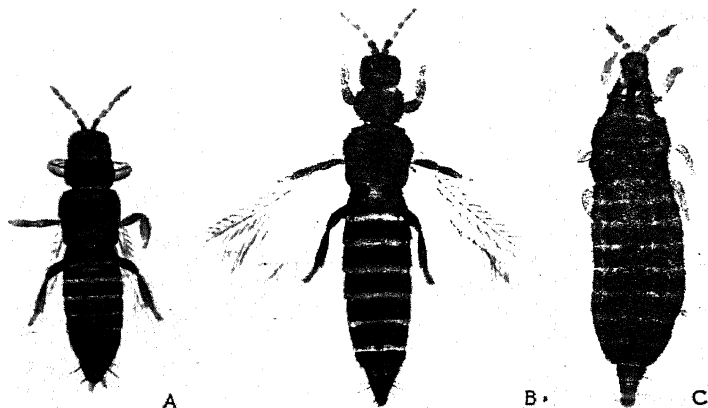


FIG. 164.—*Frankliniella lycopersici*, one of the two vectors of the spotted wilt of tomato in Australia. A, adult male; B, adult female; C, full-grown larva. Approx. 45 X. (Photograph furnished by J. G. Bald.)

hatching. The larval period lasts from 6 to 17 days and includes two instars. A prepupal and pupal stage of 1 to 2 weeks is normally spent in the soil, after which the adult emerges, and the life cycle which requires a period of about 35 days is thus completed. The activity of the thrips is greatly increased by warm weather; Bald (1937) has shown a striking influence of the temperature on the number of plants becoming infected 12 days later.

Yellow Spot of Pineapple (*Ananas Virus 1*).—Yellow spot is one of the major diseases of pineapples in the Hawaiian Islands. It was first thoroughly defined and described by Illingworth in 1931, but the cause of the disease was not known definitely at

that time. The virus nature of the disease and its method of spread were determined definitely by Linford in 1932. The disease was first observed in a small area in Hawaii in 1926. In subsequent years, it spread rapidly throughout the islands. Yellow spot was reported in the Philippines in 1935 (Serrano 1935).

The first symptom of yellow spot is the so-called "initial spot," a small ($\frac{1}{8}$ to $\frac{1}{2}$ inch), slightly raised, yellowish spot on the upper surface of a leaf. It has a dark center surrounded by a

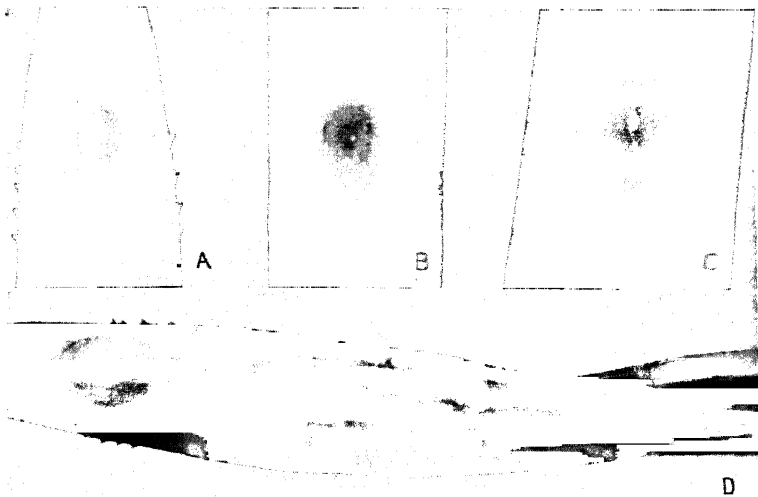


FIG. 165.—Pineapple yellow spot. A, B, and C, "initial spots," or primary symptoms; D, more advanced symptoms involving a long chlorotic streak and a large area of necrosis at its base. The original initial spot is seen at the left near the apex of the leaf. (After Linford.)

halo of yellow (Fig. 165). The spot is usually found 3 to 8 inches from the base of the leaf, its center often marking the oviposition puncture of the vector (*Thrips tabaci* Lind.). A yellow streak eventually appears, extending from the spot to the white tissue at the base of the leaf. The streak is often constricted into small circular areas, giving a beadlike appearance. As the disease advances, the streak becomes water-soaked and finally necrotic. Similar symptoms appear progressively on the upper whorls of leaves. The tissues of the stem on the affected

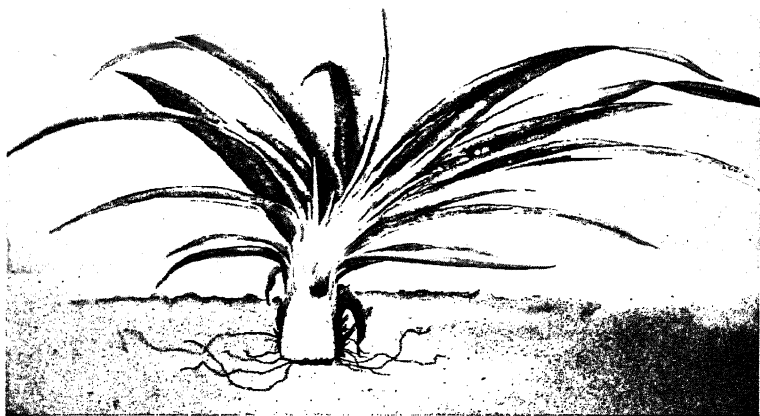


FIG. 166.—Side rot, an advanced stage of yellow spot in which extensive necrotic areas have developed along one side of the plant. (After Illingsworth.)

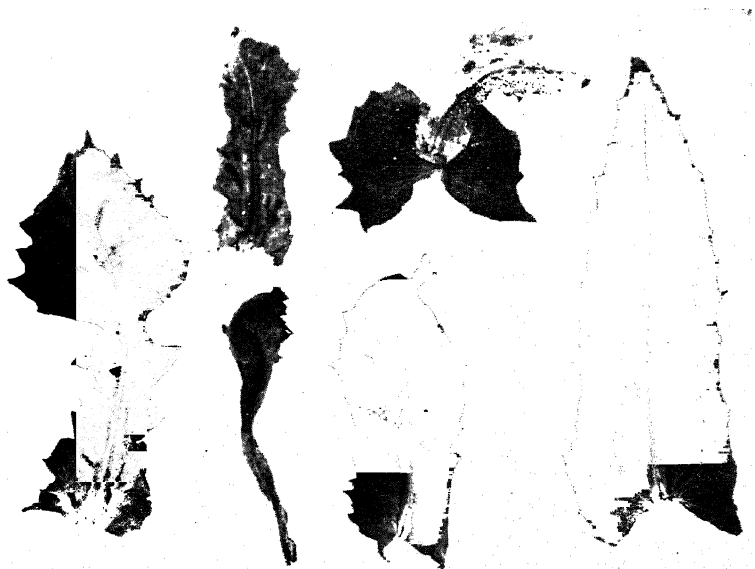


FIG. 167.—Yellow spot of pineapple on leaves of *Emilia sagittata*, a common weed in the vicinity of pineapple fields in Hawaii. (After Linford.)

side cease to elongate so that the axis becomes curved toward the affected side. Finally, the entire plant droops and dies. The distorted one-sided nature of the affected stems gave rise to the earlier name "side rot" (Fig. 166).

Several weeds growing in the vicinity of pineapple fields are affected by the virus of yellow spot. Symptoms of various types are produced on these weeds (Fig. 167). The most prevalent weed host is *Emilia sagittata* (Vahl.) DC., which is also a preferred host of *T. tabaci* Lind. and has been shown to be the principal

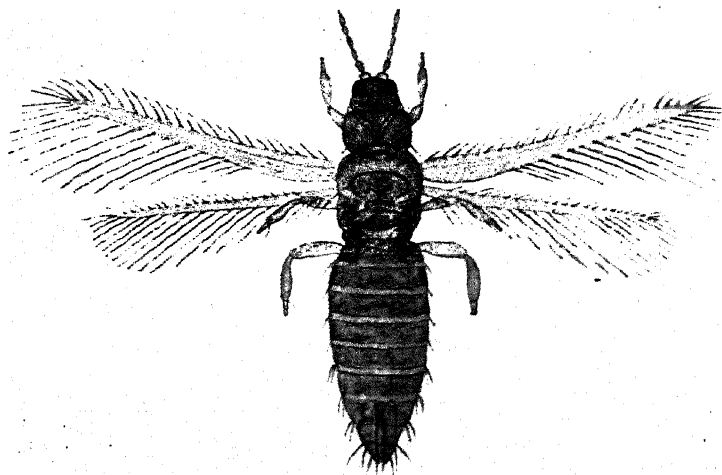


FIG. 168.—*Thrips tabaci*, the vector of pineapple yellow spot. (After Chittenden.)

source of infection on pineapples. Yellow spot has much in common with spotted wilt of tomato and other plants, and it is possible that the two diseases are caused by the same virus or closely related viruses.

Transmission of Yellow Spot.—The disease is transmitted by *Thrips tabaci* Lind. (Fig. 168), the only known vector. The virus is not transmitted readily by artificial inoculation although it can be transmitted in this way (Carter 1935). An incubation period of approximately ten days in the insect is required. As in the case of spotted wilt, the vector is unable to acquire the virus in the adult stage by feeding on infected plants. The virus is

acquired in the larval stage and survives pupation, the adults that develop from viruliferous larvae being also viruliferous. The incubation period of the virus in pineapple and *Emilia sagittata* varies from 7 to 15 days.

Thrips tabaci is an introduced species in Hawaii, but it is well distributed throughout the Hawaiian Islands. It has a wide host range, having been collected from 66 different species of plants of which *E. sagittata* is one of the most important. Its life history has been studied by Sakimura (1932).

In Hawaii, *T. tabaci* reproduces parthenogenetically throughout the year, and sexual reproduction is rare. There are two larval instars, a pupal and prepupal stage, that require approximately two weeks for completion. The eggs hatch in 4 to 5 days, and the average adult life is 32.4 days. The rate of development is slower in the cooler months, but the insect does not hibernate.

Control of yellow spot by controlling the vector, although offering promise, has not been very effective. Biological control through imported parasites (*Thripoctenus russelli* Crawford and *T. brui* Vuillet) has been attempted (Sakimura 1937), as well as control by dusting with nicotine (Carter 1932).

5. TRANSMISSION BY LACE BUGS; BIOLOGICAL AND SPECIFIC

Leaf Curl (Kräuselkrankheit) of Sugar Beet (Beta Virus 3).—This is a destructive disease of sugar beets in local regions of Germany and Poland. It is similar in many respects to the curly-top disease of beets in America but apparently is not identical with it. For many years, the disease had been associated with infestations of the beet leaf bug (*Piesma quadrata* Fieb.), but it was thought to be due to the toxic effect of the insect (Dyckerhoff 1927). It is known now, however, that the disease is caused by a virus disseminated by *P. quadrata*.

The symptoms of leaf curl differ in several respects from those of curly top. Willie (1928) recognized primary symptoms that develop shortly after the insects feed upon the plant. These consist of light-colored spots marking the feeding punctures, a reduced turgor of the leaves, and occasionally premature death of the young plants. The secondary symptoms develop after a latent period that varies with numerous influencing factors. They consist of a swelling and clearing of the veins, accompanied

by a distortion of the leaf. The petioles bend inward, forming a close compact "head-lettuce" type of growth. The plants are badly stunted, and the older leaves wither and die, forming a conical-shaped crown. The sugar content of the roots is greatly reduced, and the diseased plants often die prematurely.

The host range has not been studied extensively, but it is known to include red beets, mangold, spinach, and *Rumex acetosa* (Wille 1929). Wild-growing Chenopodiaceae as a rule



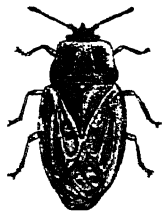
FIG. 169.—Savoy of sugar beet. Note the curled and crinkled condition of the younger leaves. (Photograph reproduced by courtesy of Division of Sugar Plant Investigations, U. S. Dept. Agr.)

do not develop the secondary symptoms of the disease and, because they are all annual plants, are of no significance in overwintering the virus.

Transmission of Leaf Curl.—The lace bug (*Piesma quadrata* Fieb.), is the only known vector of the disease. Leaf curl has been transmitted by artificial sap inoculation but in only a small percentage of trials. It is not transmitted through the seed of sugar beets. Nymphs of the vector are not capable of transmitting the disease, although adults bred from nymphs fed on

infected plants are viruliferous. The effectiveness of transmission and the severity of the symptoms caused depend to some extent on the number of insects and the time of feeding. Ten insects feeding for 2 hours are as effective as 3 insects feeding for 24 hours. One insect feeding for 24 hours or 10 insects feeding for 1 hour cause only mild symptoms. The virus is not transmitted congenitally in the eggs of the vector. The individuals of each new generation must acquire the virus by feeding on infected plants.

The virus may overwinter either in infected plants or in the adult insects. The vector survives the winter as adults in a quiescent stage. They become active in the latter part of March or the first part of April. Egg laying begins in May, the eggs being deposited chiefly on the surface of the underside of the leaves. The first brood of adults appears in July. There are usually two broods a year in Germany. There are five larval instars. The control of the disease depends upon destroying the infected plants that might serve as sources of primary infection and upon control of the insect vector. The latter is accomplished with some success by destroying the cover along the drainage ditches in which the adults hibernate and by the use of trap rows of early planted beets on which the insects are killed by spraying.



I

FIG. 170.—*Piesma cinerea*, the vector of savoy of sugar beet. (After Riley.)

“Savoy” of Beets (Beta Virus 5).—“Savoy,” a virus disease of sugar beets and garden beets, was reported in the United States in 1937 by Coons, Kotila, and Stewart. This disease is very similar to the *Kräuselkrankheit* of Germany and is transmitted by *Piesma cinerea*. Coons, Kotila, and Stewart, however, state that it is distinctly different from both *Kräuselkrankheit* and curly top.

Affected plants have dwarfed, down-curved, savoyed leaves, and the symptoms are especially pronounced on the innermost leaves (Fig. 169). Primary symptoms are vein clearing and a thickening of the veinlets, giving a netted appearance to the dorsal leaf surface. In later stages, the roots are discolored, and the phloem is necrotic. The sugar content of affected roots is lowered.

Piesma cinerea (Fig. 170) is the only known vector. Viruliferous and nonviruliferous individuals are found. Savoy is not transmitted by *Myzus persicae*, *Aphis rumicis*, or *Eutettix tenellus*. The disease has not been transmitted by artificial sap inoculation. The latent period in the sugar beet is 3 to 4 weeks. The virus overwinters in affected beet plants and in the vector. The host range of the disease has not been determined. The economic importance of the disease is not great.

6. TRANSMISSION BY WHITE FLIES; BIOLOGICAL AND SPECIFIC

Leaf Curl of Cotton (*Gossypium Virus 1*).—Leaf curl (leaf crinkle) is a destructive virus disease of cotton in the Sudan and in Nigeria. For many years, the disease was confused with injury caused by leaf hoppers; but in 1930 Golding, working in Nigeria, and Kirkpatrick, in Sudan, independently discovered the virus nature of the disease and demonstrated its transmission by a white fly (*Bemisia gossypiperda* Misra and Lamba).

The symptoms of leaf curl vary extensively. On some hosts and under some conditions, the leaves may show only a mosaic chlorosis or a downward curl of the leaf margins, but a definite crinkle produced by the "net-vein enations" is the most characteristic symptom on Sakel cotton. In all cases, the plants are stunted, and the yields are reduced.

A similar disease of tobacco occurs in South Africa, Java, and Rhodesia. In Java, it is known as *kroepeck*. Storey (1931) and Thung (1932) have shown that *kroepeck* is transmitted by a species of *Bemisia*. Storey (1932b) concludes that *kroepeck* of tobacco is identical with leaf curl of cotton and is spread by the same vector (*B. gossypiperda*). Smith (1937a), however, lists these two viruses as being different.

Transmission of Leaf Curl.—The cotton white fly (*Bemisia gossypiperda*) is the only known vector of the disease (Kirkpatrick 1931). Aphids and leaf hoppers have been tested but with negative results. The disease is not transmitted by artificial sap inoculation. All evidence indicates that the virus is not transmitted through the seed, and there is no evidence of transmission through the soil.

The latent period of the disease in the cotton plant varies from 8 to 34 days but in the majority of cases is between 11 and 19

days. The vector may acquire the virus by feeding on an infected plant 3 hours or less, and if an incubation period in the insect is required it is not longer than 30 minutes. If the larvae have fed on infected plants, the adults are viruliferous without again feeding on infected plants. Viruliferous insects retain the virus for their entire lifetime. The virus is not congenitally transmitted through the eggs of the vector. The cotton white fly



FIG. 171.—Cassava mosaic. A leaf showing typical symptoms of a severely affected plant. (After Storey and Nichols.)

was described as a new species by Misra and Lamba (1929), and a few brief notes on its habits and life history were given.

The virus of cotton leaf curl apparently survives the “dead” season in the ratoon cotton which is nearly always infective when new growth is made. Control of leaf curl will depend upon the control of the white fly or the elimination of sources of primary infection. Rotations planned with the view of eliminating the ratoon cotton are being tried.

Cassava Mosaic (*Manihot Virus* 1).—A mosaic disease of cassava (*Manihot utilissima* Pohl) has been known in East Africa since 1894 and is now known to occur throughout Western and

Central Africa where it incurs much loss. The disease is a typical mosaic disease although the symptoms vary widely. The leaves are mottled with distinct chlorotic areas and often are badly distorted (Fig. 171). Severely affected plants are stunted in proportion to the degree of leaf chlorosis. There are several strains of the virus, some producing much milder symptoms than others.

Transmission of Cassava Mosaic.—The virus of this disease apparently is not transmitted by artificial sap inoculation but is transmitted readily by grafting and by vegetative propagation. It is not transmitted through the true seed. One or more species of white flies (*Bemisia spp.*) are effective vectors of cassava mosaic. Insect transmission was first demonstrated by Ghesquiere (1932) and confirmed by Storey (1934) and Golding (1936). More complete studies of insect transmission have been reported by Storey and Nichols (1938). There is some question of the identity of the vector. It has been referred to as a variety of *B. gossipiperda* (var. *mosaicivectura*) but one authority has identified specimens as a species closely related to *B. nigeriensis* Corb. (Golding 1936). Perhaps more than one species may be involved.

The vector is capable of transmitting the virus only by feeding on immature leaves, less than one-fourth their full length. A minimum latent period of the virus in the plant of 12 to 20 days is required. The incubation period of the virus in the insect vector is not known. Successful transmission experiments have all involved the use of large numbers of insects, and it is not known whether or not a single insect can transmit the virus.

7. TRANSMISSION BY MITES

"Reversion" of Black Currants (Ribes Virus 1).—A disease of considerable economic importance affecting black currants in England and Western Europe is known as "reversion" (Fig. 172). The name was applied by growers and practical horticulturists because affected plants appear to revert to the wild type from which the improved cultivated varieties were derived. The symptoms are not clear-cut, and the disease has been confused with a number of other abnormal conditions. The most characteristic diagnostic symptoms are found on the leaves of the succulent vegetative shoots developing from buds formed the previous year near the top of the season's growth. The central

lobes of these leaves have a smaller number of submain veins and are more coarsely dentate than normal leaves. Reverted plants fruit very sparsely and eventually may become completely sterile (Amos and Hatton 1927).

Reversion has been studied most extensively in England where it is regarded as a major disease of black currants. The English



FIG. 172.—Reversion of currants. The branch on the left is affected, the one on the right is normal. (Photograph reproduced by permission of East Malling Research Station, Kent, England.)

workers have presented strong evidence that the disease is caused by a virus. Because of the nature of the disease, its study has met with many difficulties, and it has not been easy to reach final conclusions in respect to many phases of the disease.

Inasmuch as the symptoms of reversion resembled genetic mutations associated with chromosome abnormalities, Darlington

(1927) made a study of the chromosome complements in somatic cells from normal and reverted plants. The chromosome conditions in both plants were found to be essentially the same, and it was concluded that the possibility of a genetic explanation of the cause of reversion was remote.

Transmission of Reversion.—Reversion as it occurs in nature is closely associated with a condition known as "big bud" caused by the mite (*Eriophyes ribis*). These mites live in the buds, inhibiting their normal expansion and causing them to swell. The buds are eventually killed, and the mites then migrate to new buds which they infest (Massee 1927). Evidence has been presented (Amos, Hatton, Knight, and Massee 1927) to show that this mite is also the vector of reversion. Lees (1925), on

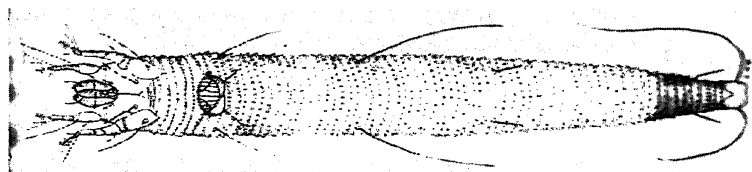


FIG. 173.—*Eriophyes ribis*, the vector of reversion of currants. Very highly magnified. (Drawing reproduced by courtesy of East Malling Research Station, Kent, England.)

the other hand, although recognizing the possibility of mite transmission, maintains that it can be transmitted in nature independently of the presence of the mite. Big bud is not believed to be a symptom of reversion; the two conditions are thought to be distinctly different. Big bud may occur on bushes that show no symptoms of reversion, and the disease is occasionally found on bushes that are free of big bud. In experiments in which buds were artificially infested with mites from reverted plants, 90 per cent of the successfully infested plants developed reversion and 92 per cent of the control plants not infested with mites remained healthy. Because of certain difficulties in working with the mites, the results are not conclusive beyond question, but there is strong circumstantial evidence supporting the view that reversion is transmitted by the mites. If this conclusion is proved to be correct, this is the first and only virus disease of plants known to be transmitted by a mite.

The disease is definitely infectious and is transmitted by grafting but has not been transmitted by artificial sap inoculation. However, the appearance of reversion on a number of stocks, grafted with reverted scions that did not make a successful union, led Lees (1925) to conclude that the virus was transmitted by contact. The disease is not transmitted by the seed. The incomplete and often contradictory nature of much of the data dealing with the transmission of this disease makes it difficult to reach a satisfactory conclusion concerning its nature and methods of transmission.

The natural spread of the disease in the field has been studied by Amos and Hatton (1928), who showed that a rapid spread could be traced to a center of exceptionally heavy infestation of the big-bud mite and reversion. Careful roguing and the selection of healthy plants for cuttings give promise of successful control of the disease.

Eriophyes ribis, the black-currant-gall mite, is a very small mite with a relatively long cylindrical body (Fig. 173). The females measure only 220 microns in length, and the males are about 150 microns long. They feed upon several species of *Ribes* but appear to be confined to plants of that genus. The mites leave the old buds in late spring, and in the latter part of May they enter the new buds and make their way to the center where they breed. Eggs may be found in the buds the year round and are deposited, also, on the leaves and in the blossoms. They hatch within 3 to 7 days. Infested buds begin to swell in late summer, and the following spring they fail to open and usually die. The mites are disseminated from bush to bush by insects and by wind.

8. NATURE OF TRANSMISSION OBSCURE

Wheat Mosaic (*Triticum* Viruses 1 to 7).—In 1919, a destructive disease of winter wheat was observed in Illinois and Indiana (McKinney 1923, 1937). The true nature of the disease was not recognized at that time, but further investigations have shown the condition to be a complex one caused by several closely related viruses (McKinney 1937). One or more of the viruses have been reported from Illinois, Indiana, Kansas, Nebraska, Maryland, North Carolina, and Virginia. The viruses affect certain varieties of wheat, barley, rye, emmer,

spelt, and einkorn. They are most destructive on certain varieties of winter wheat. Spring wheat is rarely affected although it is susceptible if planted in the autumn in a region of mild winters. The diseases have not caused a great economic loss partly because of their limited distribution and partly because of the availability of suitable resistant varieties of winter wheat. However, they are potentially very destructive because entire fields may be made worthless in regions where the diseases are prevalent.

The symptoms caused by the viruses vary greatly with the strain or strains involved, the variety of suscept. and environmental conditions. One of the principal effects is a dwarfing that results in a rosette type of growth that does not head out. Various degrees of chlorosis in a variety of patterns are often observed, but chlorosis is not universally present on affected plants.

Transmission of Wheat Mosaic.—The viruses of wheat mosaic are transmitted readily by artificial sap inoculation, but the methods of transmission in nature are not definitely known. These viruses are unique in that they appear to be transmitted in nature only through the soil (Webb 1927, 1928). Although it is possible that aphids may transmit at least one of the viruses (Haskell and Wood 1923), insects that feed upon the aerial parts of the plant apparently are not important in natural transmission. The viruses are not transmitted through the seed, but the disease develops in severe form when seed are planted in infested soil in insectproof cages. Plants grown in sterilized soil or in non-infested soil do not become diseased although they are not protected from insects. The viruses may survive indefinitely in the soil and cannot be removed by extensively washing the soil. Infection takes place through the roots and through the crown of the plant, but the mechanism of ingression is not known. This aspect of the problem needs further study.

Latent (X) Virus of Potato (Solanum Virus 1).—This unique virus was discovered by Johnson (1925), who inoculated tobacco plants with juice of potato plants that appeared to be free of virus infection and found that distinct virus symptoms were produced on the inoculated tobacco plants. Further experiments showed that practically all the commercially grown American varieties and many European varieties, however,

develop characteristic symptoms when inoculated with the virus. Several other species of solanaceous plant also are susceptible to the virus.

Transmission of the X Virus.—Although this virus is readily transmitted by artificial sap inoculation, it is not transmitted by aphids or other common insect vectors, nor is it transmitted through the true seeds of potatoes. Its widespread occurrence in nature, however, indicates that it must spread rapidly, but the method of transmission is still unknown. The universal occurrence of this virus in the older American potato varieties may be explained in part by the fact that the virus produces no injury to the plants and is perpetuated through the tubers. If the virus causes no injury that decreases the ability of infected plants to compete with healthy ones, it would be expected that, in time, the vegetative progeny would soon approach the condition of universal infection.

Smith (1937) has suggested that this virus may be transmitted by a flower-feeding species of thrips, but there is no record of the possibility having been thoroughly investigated. Loughnane and Murphy (1938) have presented evidence to show that the virus may be transmitted from plant to plant by leaf contact when subjected to the action of an oscillating electric fan. These authors are of the opinion that the virus spreads in nature by leaf contact, aided by the action of the wind. Cockerham (1937) found no correlation between the flowering habit of the potato varieties grown in Scotland and the presence of the X virus. It was observed, however, that the varieties found to be free of the X virus were those which reacted by severe lethal necrosis when inoculated with it. Infected individuals of these varieties would be eliminated, only the virus-free plants surviving. Thus the virus is said to be self-eliminating in those varieties which react in this way to it.

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CHAPTER X

INSECTS AND PHYTOPATHOGENIC PROTOZOA

Protozoa are single-celled animals without true organs and tissues. There are innumerable species, of widely varying size, morphology, and life habits. Many species are harmless or even beneficial, but some have been recognized for many years as the cause of fatal or severe diseases of man and other animals. Such devastating diseases as malaria, sleeping sickness, and dysentery are caused by protozoa. Insects or other arthropods have proved to be the principal vectors of a relatively large percentage of the protozoa that are pathogenic to animals, and the suppression of epidemics of these diseases has hinged largely upon the control of the arthropod vector. Plants, on the other hand, are comparatively free of protozoal diseases. In fact, it has been known for only a relatively short time that there are protozoa parasitic on plants.

Protozoa in Laticiferous Plants.—In 1909, Lafont discovered a flagellate inhabiting the latex of three species of *Euphorbia*. He described and named the organism *Leptomonas davidi*. This discovery created much interest among protozoologists and those concerned with human and animal diseases caused by flagellates. In the next few years, the same or similar species were reported from a large number of laticiferous plants in various parts of the world, mostly in tropical or semitropical regions (Franca 1911–1922, Franchini 1922–1923, Migone 1916, and others). By 1925, flagellates had been reported in more than 50 species in six or more families of laticiferous plants. Partly because few students of plant pathology were trained in protozoology and partly because of the lack of economic importance of the plant-parasitic protozoa, these organisms have received very little attention from plant pathologists. Brief reviews of the literature dealing with endophytic protozoa appeared in *Zeitschrift für Pflanzenkrankheiten* (Nieschulz 1922) and in *Phytopathology* in 1925 (Bensaude).

The protozoa found in the latex of plants are, for the most part, free-swimming flagellates. They are usually pleomorphic, the typical form being approximately 15 to 20 microns in length (Fig. 174). Multiplication, as most frequently observed, is by longitudinal binary cleavage. The protozoa are not easily cultivated on artificial media although certain species have been cultured successfully. Nearly all species found in plants have been found also in the intestinal tract of some plant-feeding Hemipterous insect. There are many similar species that have been found only in the intestinal tracts of insects. It has been suggested that probably all the endophytic protozoa were originally members of the intestinal fauna of insects and that they have adapted themselves to life in the plants on which the insects feed. Such progressive adaptation appears to be a char-

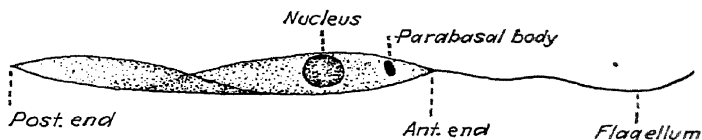


FIG. 174.—A flagellate from the latex cells of milkweed. (After Holmes.)

acteristic of this group of protozoa. Referring to the order Protomastigida, which includes these flagellates, Calkins (1933) states that they "well illustrate the power of continued adaptation to new conditions in the host, leading to progressive parasitism whereby an original commensal may become a lethal parasite."

There has been some question as to the pathogenicity of the protozoa found in laticiferous plants. Lafont (1909) and Franca (1920) claimed that *L. davidi* Lafont caused a real disease of affected euphorbias. They state that infected plants may be recognized easily by a generally starved appearance followed by a yellowing and final wilting of the leaves. Infection is often localized in a single branch, which is stunted and much shorter than the normal ones. Strong (1924) also has reported striking symptoms of infection on species of *Euphorbia*, and more recently Sassuschin (1933) made similar claims for the flagellates (*L. davidi*) on *E. uralense* in southeastern Russia. On the other hand, Bouet and Rouband (1911), Rodhain and Bequaert (1911),

and Noc and Stévenel (1911) have maintained that it is difficult or impossible to distinguish infected from noninfected plants.

Migone (1916) described a flagellate from milkweed (*Araujia angustifolia*) in Paraguay and named it *L. elmassiani*. All the plants growing in one region were uniformly infected and showed no signs of injury. Four years later Franca (1921) described a new species of protozoa (*L. bordasi*), isolated by Migone from a different milkweed (*Morrenia odorata*), and in this case also no injury was evident. Holmes (1925a) likewise concluded that *Herpetomonas* (*Leptomonas*) *elmassiani* (Migone)¹ in milkweed (*Asclepias syriaca* L.) in North America was not pathogenic. It is entirely possible that these contrasting opinions are, to some extent, caused by differences in environmental factors in different localities; but in any case, it is evident that the flagellates in laticiferous plants in general cause very little injury and are not of great economic importance. However, the phenomenon is of general biological interest from several viewpoints.

The relative lack of virulence of the flagellates in laticiferous plants can be explained in part by the nature of the latex ducts that they inhabit. There are two distinctly different types of laticiferous duct, latex vessels and latex cells (Fig. 175). Both types consist of single living cells containing many nuclei and extensive vacuoles in which the latex is found. The latex is secreted by the cytoplasm and accumulates in the vacuoles. A latex vessel arises by the dissolution of the end walls of a row of meristematic cells to form a long multinucleate vessel. A latex cell results from the elongation of a single cell, which may

¹ There has been some confusion in the use of the generic names applied to the endophytic protozoa. Lafont used the name *Leptomonas* because the flagellates he observed in the latex of plants agreed morphologically with the genus *Leptomonas* Kent, all the previously known species of which were confined to the bodies of invertebrate animals. Holmes (1925b) has used the generic name *Herpetomonas* because "the genera *Leptomonas* and *Herpetomonas* were united in 1884 by Butschli, the first reviser, under the name of *Herpetomonas*." Some authorities on protozoology (Wenyon 1926), however, continue to recognize both of these genera but place the plant-inhabiting flagellates in a third genus *Phytomonas*, created by Donovan in 1909. The genus *Phytomonas* Donovan is defined by Wenyon (1926) as "flagellates which resemble those of the genus *Leptomonas*, but differ in having both an invertebrate and a plant host." Further confusion has been caused by the use of the name *Phytomonas* for a genus of phytopathogenic bacteria (Bergey *et al.* 1923).

grow and branch like a fungus mycelium. Latex vessels may unite to form an extensive anastomosing system, but latex cells occur as independent units. Most of the species of Euphorbiaceae and Asclepiadaceae have latex cells rather than latex vessels. Apparently the flagellates are confined to the latex cells and are unable to penetrate the cell walls or to parasitize the other plant tissues. Not only are they confined to the latex

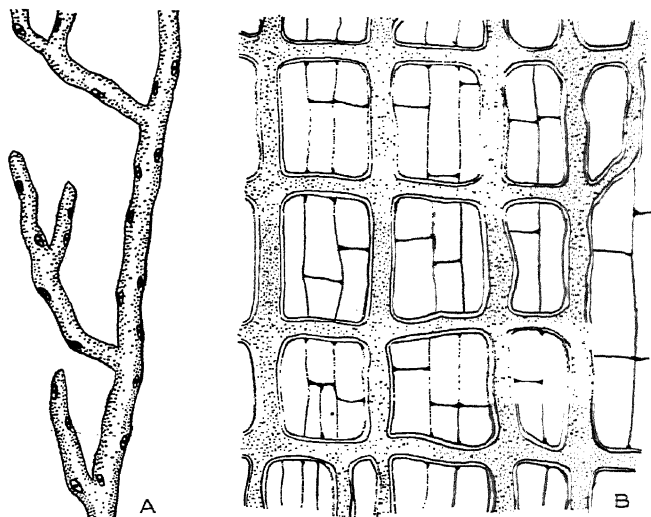


FIG. 175.—Diagrammatic representation of two types of latex duct. A, a latex cell (each cell is multinucleate, and although it may ramify extensively it is a single cell); B, a latex vessel formed by the anastomosing of a number of individual cells. Some laticiferous plants have latex cells, and others have latex vessels. Endophytic protozoa have been found chiefly in latex cells. (After Holmes.)

cells but they occur only in the vacuoles. The latex cells are limited in extent; and each cell, although it may be extensively ramified, is usually independent of the others. For this reason, the flagellates are localized so that they do not cause a systemic infection of the vital tissues. A systemic infection of the vascular elements would be likely to be much more injurious to the plants.

When the protozoa were first discovered in plants, it was observed that they were very similar in morphology to flagellates

previously found in the intestinal tracts of numerous insects, a fact which suggested that they were insect-transmitted. Lafont (1911) experimentally transmitted the flagellate (*L. davidi*) to plants by means of *Nysius euphorbiae* Hovarth, a Hemipterous insect that feeds exclusively on Euphorbiae. Bouet and Rou-

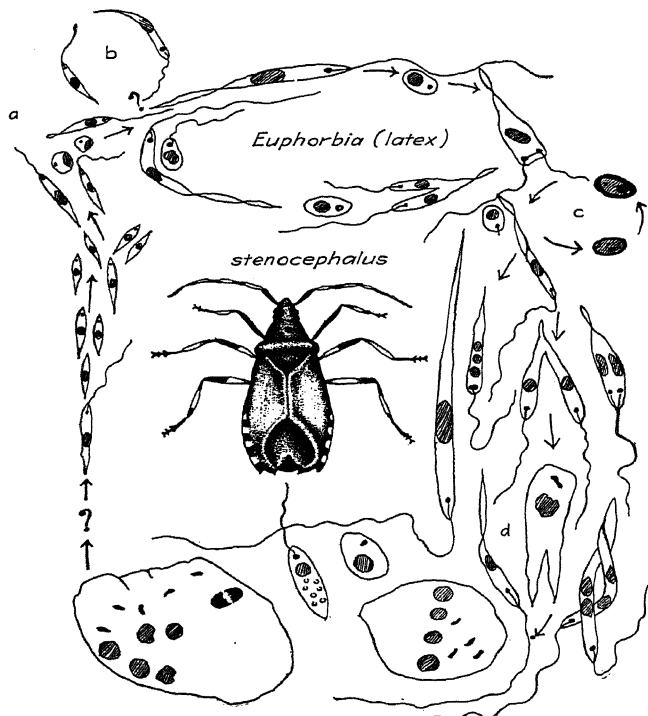


FIG. 176.—The life cycle of *Leptomonas davidi* and its vector (*Stenocephalus agilis*), according to Franca. a, infective forms; b, forms found in the fruits of Euphorbia; c, encysted forms in feces of the insect; d, forms found in the intestinal tract of the insect. (After Franca.)

band (1911) transmitted the flagellate to plants with *Dieuches humilis* Reut., another Hemipterous insect. Franca (1920) showed that *Stenocephalus agilis* (Fig. 176) was the chief vector of *L. davidi* in Portugal.

Franca (1920) was able to transmit the flagellates from plant to plant with *S. agilis* as found naturally infected in nature.

He observed flagellates in the intestinal tracts of the insects feeding on infected plants, whereas those feeding on noninfected plants were free from protozoa. By means of feeding experiments, he was able to show that the flagellates found in the insect were identical with those in the latex of infected plants. He studied also the complete life cycle of the organism as it occurred in both insect and plant hosts, describing what he interpreted as a true cyclic development comparable with that of certain insect-transmitted protozoa parasites of animals. Insects obtained from noninfected plants were fed on infected plants, and at frequent intervals specimens of the insect were killed and examined. During the first 3 days, the flagellates found in the intestinal canal of the insect were in active division and were very similar to the forms observed in the plant tissues. After the third day, multinucleated forms were observed, and these were interpreted as having been formed by isogamic conjugation. After 8 days, very small forms were found throughout the intestinal tract and in the salivary glands. These were often without a flagellum and varied in size from 4.5 to 7 microns long by 0.8 to 1.5 microns wide, whereas the forms found in plants are from 16 to 20 microns long and 1.5 to 2 microns wide. The small "metacyclic" forms are considered by Franca as the infective stage in the life cycle. The complete life cycle of *L. davidi*, as interpreted by Franca, is illustrated in Fig. 176. This is the most complete study that has been reported on the association of the endophytic protozoa with insect vectors. The evidence presented by Franca of a true cyclic development in the insect vector is in need of further study. Considering the numerous reports of endophytic protozoa in many parts of the world, it is unfortunate that more intensive studies of methods of transmission in different localities have not been made. Many of the known insect vectors are active only at night, and it is possible the nocturnal habits of vectors may explain why they have been overlooked in so many cases.

Strong (1924) has described a flagellate (probably *L. davidi*) found in Euphorbia in Central America. He showed that it was transmitted by *Chariesterus cuspidatus*, a coreid bug, and demonstrated it in all parts of the intestine and in the salivary glands of the vector. The organism manifested a greater diversity of form in the insect than in the latex of the plants.

Forms with the posterior end twisted in a helicoid manner, small round or oval forms, with or without a flagellum, and typical cystic forms were observed. Slender, short forms with a rigid flagellum were the most common ones in the salivary glands. Reproduction through binary fission was demonstrated in the insect, but no multiple fission was observed.

Holmes (1924, 1925*a*, *b*, *c*) found *Oncopeltus fasciatus* (Dall), a red-and-black Hemipterous insect, closely associated with milkweeds infected with *Herpetomonas elmassiani* (Migone) and considered it as the most probable vector. The geographical distribution of the insect vector coincided very closely with the distribution of infected milkweeds (Holmes 1925*b*). Both adults and nymphs contained flagellates in their salivary glands, but the flagellates were not observed in the intestinal tracts of any of the specimens examined. These flagellates were similar to ones found in the plants in all respects except size. Those found in the insects were somewhat larger than those in the plants, but the smaller individuals found in the insects were well within the range of those in the plants. A characteristic twist of the body was present in flagellates from both sources. It was concluded, therefore, that the same species was involved in both cases. No cyclic changes of the protozoa in the insect body were reported.

The salivary gland of *O. fasciatus* is a three-lobed gland located in the thorax and emptying through a single efferent duct. The flagellates were very abundant in the most dorsal lobe, and a few were found in the anterior lobe, but the posterior lobe was always free of them. It is not known how the salivary gland becomes infected, for the protozoa were found neither in the alimentary canal nor in the blood cavity.

DuPorte (1924, 1925) reported the presence of protozoa in the latex of the following plants: *Asclepias syriaca*, *Convolvulus sepium*, *Norus alba*, *Lactuca canadensis*, *L. sativa*, *Apocynum* sp., *Hieracium aurantiacum*, *Taraxacum officinale*, *Ficus* sp., *Sonchus* spp., *Euphorbia* sp., and *Chelidonium majus*. No descriptions were given, although both flagellates and amoeba were mentioned. No insect vectors were reported. In two cases, *A. syriaca* and *C. sepium*, it was stated that the protozoa were transmitted through the seed of infected plants.

Noguchi and Tilden (1926) have reported comparative studies of flagellates from insects and plants. These authors described

and named two species from bugs feeding on laticiferous plants and from the plants as follows: *Herpetomonas oncopelti* from *Oncopeltus fasciatus* (probably the same as the one described by Holmes from the same insect as *H. elmassiani* Migone), and *H. lygaeorum* from *Lygaeus kalmii*, a bug closely related to *Oncopeltus*. These authors compared these flagellates and others obtained from latex of plants and from other insects with the *Leishmania* forms that are parasitic on higher animals and found them to be quite different.

Franchini (1922*d*) has described a flagellate infection of cabbage, a nonlaticiferous plant. The plants were infested with pentatomid bugs (*Pentatoma omatum* and *P. oleraceum*), and the affected leaves were yellow and wilted. Smears made from the leaves and the intestines of the insect revealed the presence of similar flagellates. However, the evidence that the protozoa were true parasites within the tissues of the cabbage is not convincing. Franchini (1923*d*) claims to have inoculated *Euphorbia* plants successfully with flagellates from the intestines of several species of fly, but the methods of infection in nature and the life cycles of the protozoa were not determined. However, latex was recognized in the intestinal tracts of flies and it was suggested that the feces of flies feeding on droplets of latex secreted by the corolla of the flowers might account for natural infection. Many of the reports of Franchini have been fragmentary, containing very little experimental evidence, and later work supporting his conclusions has not been reported.

Some studies have been made to determine if the plant-inhabiting flagellates are capable of infecting vertebrates. Strong (1924) has shown that in Central America a bug, [*Charisterus cuspidatus* (Distant)] transmits the flagellates of *Euphorbia* and that the bugs are eaten by a lizard (*Cnemidophorus lemniscatus*). In the posterior portion of the intestines of the lizard a flagellate indistinguishable from the insect flagellate was found. These were inoculated into monkeys, dogs, guinea pigs, and mice; but only one lesion was obtained, on the monkey, in which flagellates of the *Leishmania* type were present. The ulcer and the flagellates resembled somewhat the lesions of a cutaneous leishmaniasis prevalent in the locality. The evidence, however, is not sufficient to justify the conclusion that the endophytic protozoa are the cause of the disease affecting man.

Phloem Necrosis of Coffee.—Even though the flagellates found in laticiferous plants are not highly pathogenic, protozoa cannot be ignored as plant pathogens. Stahel (1931, 1932, 1933, 1934) has shown that the destructive phloem necrosis of the coffee tree in Dutch Guiana (Surinam) is caused by a flagellate which he named *Phytomonas leptovasorum*. According to



FIG. 177.—A coffee tree affected with phloem necrosis. (After Stahel.)

Stahel, phloem necrosis is the most destructive disease of coffee in Surinam. The disease has been known for more than 30 years, but its nature and cause were not understood. It occurs in two forms, one acute and the other chronic; but all intergrading types may be found. The chronic form is the most common and is characterized by a premature yellowing and dropping of the leaves. New leaves are put out, but they are smaller and fewer than the first ones. Finally, after a period varying from

3 to 12 months, the entire tree dies (Fig. 177). In the acute form the leaves turn yellow and within 1 or 2 days begin to wilt, and the entire tree will usually die within 1 to 2 months. This type is most frequent in the beginning of a dry period but may occur at other times.

As the name of the disease implies, the seat of the trouble is in the phloem tissues which become necrotic while the adjacent vascular tissue is discolored by impregnation with wound gum. Preceding the appearance of necrosis, many of the sieve tubes undergo multiple division, a condition giving rise to many abnormally small sieve tubes. The necrotic sieve tubes always contain large numbers of flagellates resembling in many respects those found in the latex of euphorbias and other laticiferous plants (Fig. 178). Although microscopic examination of living material and stained preparations leave no doubt as to the protozoal nature of the pathogenic organisms, Stahel has not been able to culture them in artificial media.

The method of transmission of phloem necrosis is not definitely known, but much circumstantial evidence indicates that an insect vector is responsible. In 1935, Bünzli, who made an extensive study of insects associated with the roots of the coffee plant, concluded that a coccid (*Rhizoecus coffeae*) was the vector. This insect, which is attended by the ant (*Rhizomyra paramaribensis*), feeds upon the roots of the coffee tree, and in a limited number of uncontrolled experiments Bünzli obtained data which led him to believe that it was the agent of transmission. Stahel, however, was unable to confirm Bünzli's results and concluded that the bug (*Lincus spathuliger* Bredd¹) is the true vector, although definite proof of transmission was not obtained.

The improbability of *R. coffeae* serving as a vector is indicated by the fact that the salivary canal of its mouth parts is too small to permit the flagellates to pass, being only $\frac{1}{4}$ micron in diameter, whereas the flagellates are 1 to $1\frac{1}{2}$ microns thick. *Lincus spathuliger*, however, has a salivary canal measuring 4 microns in diameter. This Hemipterous insect is also attended by ants (*Pheidole biconstricta*), feeds on the tap root near the surface of the ground, and is potentially an effective vector.

¹ Originally referred to as *L. securiger* but corrected by correspondence with the author.

In view of the fact that the flagellates of laticiferous plants are transmitted by Hemipterous insects, it seems only reasonable to suspect that similar insects may be responsible for the transmission of *P. leptosporum*. It would be difficult to account for their transmission by any other agency than insects. It is



FIG. 178.—*Phytomonas leptosporum*, the flagellate pathogen of phloem necrosis of coffee, in the sieve tubes of affected coffee plants. (After Stahel.)

hoped that further investigations will soon clear up this important phase of the problem.

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CHAPTER XI

MITES, NEMATODES, AND OTHER SMALL ANIMALS AS VECTORS OF PLANT DISEASES

In previous chapters, our discussion of vectors of plant diseases has been limited almost entirely to insects. However, there are, in addition to insects, a number of small animals that serve as effective vectors. Among these may be mentioned mites, nematodes, slugs, earthworms, and birds. A few examples of disease transmission by these animal vectors will be discussed briefly.

MITES

Mites are small, insectlike arthropods closely related to spiders and ticks. They belong to the order Acarina, which includes both mites and ticks. The principal difference between mites and ticks is one of size, the larger members of the order being known as ticks, the small ones as mites. The latter differ from insects in having typically four pairs of legs, no antennae, no true jaws or compound eyes, and no distinct body segmentations (Fig. 179). One family (Eriophyidae), however, is characterized by having only two pairs of legs (Fig. 173). The larvae of most species, immediately upon hatching, have only three pairs of legs, but they soon molt and transform into nymphs having all four pairs. After one or two additional molts, they acquire external sexual organs and assume the adult form. Mites are found in many localities and have a wide variety of habits. Some are free-living, but many are parasitic upon animals or plants, causing much economic loss. Only a few species of mite have been incriminated definitely as vectors of plant diseases, but they have not been studied extensively from this viewpoint.

An excellent review of the Acarina is given by Banks (1915), who recognizes 8 superfamilies composed of 31 families. Those mites most closely associated with plants and plant diseases are found chiefly in the families Eriophyidae and Tyroglyphidae. *Pediculopsis graminum* Reut., the vector of *Sporotrichum poae*,

causing silver top of June grass and bud rot of carnations, belongs to the family Tarsonemidae, many of which feed on growing plants and cause various deformities.

The Eriophyidae are among the smallest of mites, few of them reaching more than 250 microns in length; many are only half as long. The body is usually much elongated with a multiannulated abdomen. The adult characteristically has two pairs of legs. All species of the Eriophyidae feed on plants and usually cause galls. The forms of the galls vary; but, in general, they are characterized by the production of a velvety mass of abnormal

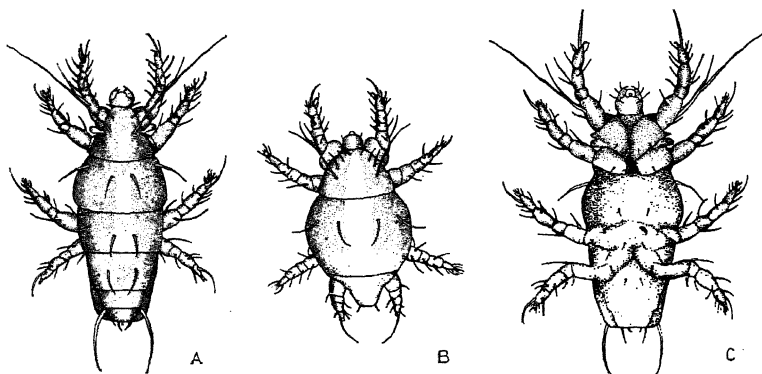


FIG. 179.—*Pediculopsis graminum* Reut. The mite that serves as a vector of bud rot of carnations. A, male dorsal view; B, male ventral view; C, nongravid female, dorsal view. (Redrawn after Stewart and Hodgkiss.)

trichomes, either exposed on the surface or enclosed in pouches or capsules formed by the leaf tissue. The term “erinese” is applied to the galls produced by these mites.

Some species inhabit buds, causing them to become abnormally large but inhibiting normal opening and growth. *Eriophyes ribis* causes big bud of currants in England and Europe, and is the best known of the bud-infesting species. It also is a vector of “reversion,” a virus disease of currants.

The Tyroglyphidae are most frequently associated with stored foods and decaying plant tissues. A few species have been incriminated as vectors of plant pathogens, and several others have been under suspicion. The family is characterized by the occurrence of a peculiar and unusual nymphal stage, the so-called

"hypopus," which is strikingly different from the other stages. The body of the hypopus is hard and tough; it has no mouth, and its legs are short and ill-adapted to walking. For many years, it was not recognized as a stage of development but was considered a distinct species. It is now known to be a stage especially adapted for ensuring distribution to new sources of food. The hypopus has, on its ventral surface, a number of suckorial disks by means of which it attaches itself to an insect (Fig. 181). Having no mouth parts, it does not feed upon the insect but merely uses it as a means of transportation. After transportation to a suitable locality, its hold on the insect is released, and the mite undergoes metamorphosis into a typical nymph and begins to feed again. Tyroglyphid mites that inhabit the bark of trees are transported by bark beetles and other bark-infesting insects. Those which live on decaying material are transported by flies and other insects that are attracted to decayed plant tissues. The British Tyroglyphidae were studied extensively by Micheal (1901, 1903), and a revision of the family was published by Banks (1906).

The Relation of Mites to the Silver Top of June Grass and Bud Rot of Carnations.—Silver top of June grass (*Poa pratensis* L.) and other grasses is a disease that causes the flower panicles to wither before they have become fully expanded. The affected parts lose their chlorophyll and become bleached and silvery in color, suggesting the name "silver top." For many years, the trouble was thought to be caused by insects, but in 1908 Stewart and Hodgkiss reported evidence from work begun in 1902 which showed that the condition was caused by a fungus disseminated and inoculated by a mite. The fungus was described and named *Sporotrichum poae* by Peck in 1903. The mite was identified as *Pediculopsis graminum* Reut., which had previously been reported by Reuter (1900) as associated with the disease in Finland. The mites (Fig. 179) crawl down the grass culms under the sheath to the base of the panicle where the tissues are relatively soft and succulent. They inoculate the tissues with the fungus which they carry with them. The mites apparently feed on the fungus and the decayed tissue that develops at the base of the panicle. Carnation bud rot is a disease characterized by an internal decay of the unopened buds resulting in the failure of the buds to expand into normal flowers (Fig. 180). On dissecting the

unopened buds the interiors are found in various stages of decay, and numerous mites always are present in the decayed tissues. Bud rot of carnations was first described in Nebraska by Wolcott in 1905. He attributed the injury entirely to mites. The following year, Heald (1906) reported the same trouble to be caused by a fungus transmitted by mites. The fungus at first was thought to be a species of *Fusarium* but was later identified as a species of *Sporotrichum*. A more complete description of the trouble was published by Heald (1908) with experimental proof of the patho-



FIG. 180.—Two carnation flowers cut open to show the internal decay associated with infestation by the mite *Pediculopsis graminum*. (Courtesy of the New York Agricultural Experiment Station, Geneva.)

genicity of the fungus and its transmission by mites. The fungus was described and named *S. anthophilum* by Peck. The mite was described and named *Pediculoides dianthophilus* by Wolcott (1908). Later in the same year, Stewart and Hodgkiss (1908) published a report of their study of the same disease in New York. These authors concluded that bud rot of carnations and silver top of June grass were caused by the same fungus (*S. poae* Peck) and that the mite was the same as that described in 1900 by Reuter as *Pediculopsis graminum*.

The evidence obtained by all workers on this disease indicates that the fungus is entirely dependent upon the mites for its introduction into the carnation buds. Whether or not the mites are dependent upon the fungus in any way has not been determined,

but the decayed tissue appears to be favorable for their development. The method of overwintering of the fungus is not known. There is no evidence to show whether the fungus is constantly associated with the mites or whether the mites become contaminated from some other source each year. The eggs are not deposited by the female mites, the young developing to maturity inside the bodies of the gravid females, which on death disintegrate to liberate numerous fully developed young. The mites probably survive the winter as adults in protected places.

As described by Heald and by Stewart and Hodgkiss, carnation bud rot was confined entirely to greenhouse carnations, but recently Reiter (1935) has reported severe outbreaks of the disease in field-grown carnations. The carnations had been planted in soil that had been in June-grass sod the previous year, and the association of the mites with the decayed buds in the field was in all respects similar to that found in greenhouses.

Mites as Vectors of the Blue-stain Fungi.—Leach, Orr, and Christensen (1934) called attention to the possible role of mites in distributing the spores of the blue-stain fungi in Norway-pine logs. It was observed that from 1 to 12 or more small mites were attached to the ventral part of the thorax or in the concave wing declivities of nearly all the bark beetles (*Ips pini* and *Ips grandicollis*) found in logs infested with blue stain. The mites (which were not identified) had obviously attached themselves to the beetles before they had emerged from the infested logs. Careful examination of the logs showed mites running about in nearly all freshly made tunnels. They were also abundant in old tunnels. These mites, which apparently belong to the Tyroglyphidae, leave the beetles when they enter a log and feed on the fungi and decomposing bark tissue. When the beetles of the new brood are ready to emerge, some of the mites attach themselves to the beetles and are thus carried to new trees or logs. Microscopic examination of the mites taken from the beetles, as well as cultures made from the mites, showed that spores of the blue-staining fungi were often disseminated by them.

The possibility of the Tyroglyphid mites serving as vectors of the Dutch elm disease has been recognized by Jacot (1934, 1936). He found three species common in the bark of elms affected with the disease. They were usually associated with the brood tunnels of elm bark beetles (*Hylurgopinus rufipes* Eich. and *Scolytus*

multistriatus Marsh.) or with those of the flat-head borer (*Saperda tridentata* Oliv.). The mites were identified as follows: *Monieziella arborea* Jac., *Histiogaster fungivorax* Jac., and *Megraniella ulmi* Jac.

The migration of these mites from tree to tree is effected chiefly through the hypopial nymph, which is more drought-resistant than the normal form and which attaches itself to any passing insect and is transported by the insect to a new location. The hypopial nymphs are incapable of feeding and do not carry spores internally but are capable of disseminating those adhering to bristles, legs, or body plates. Many of the mites were taken in bark-beetle tunnels which bore fresh coremia of *Ceratostomella ulmi*.

Fransen (1934) also called attention to mites as a factor in the transmission of *C. ulmi*. A mite *Pseudotarsonemoides innumabilis* Vitzth.) is associated with the beetles in Holland. The mites, when present, disseminated the spores within the brood galleries and stimulated more profuse sporulation in the pupal chambers, thus increasing the effectiveness of the beetles as vectors.

Mites as Vectors of Microorganisms Causing Decay of Plants. Woods (1897) described the action of the bulb mite (*Rhizoglyphus hyacinthi* Boisd.) (Fig. 181) on the Bermuda lily. The mites were found in large numbers on decaying roots and bulbs. By colonizing the mites on healthy plants, it was shown that they would attack uninjured tissues, which were soon destroyed by fungi and bacteria introduced by them. No detailed study was made of the fungi and bacteria responsible for the decay.

Hodson (1928) has more recently studied the bionomics of the same mite (referred to as *R. echinops* Fumouze & Robin). He concludes that the bulb mite is not a primary parasite of uninjured hyacinth bulbs. In an extensive series of experiments, bulbs were infested with the mites under various conditions. If the bulbs were injured mechanically or by nematode infestation, they were rapidly destroyed by the mites and associated microorganisms, whereas control plants kept free of mites were not destroyed. It was further concluded that "the mites can undoubtedly carry fungus spores from bulb to bulb."

McDaniel (1928) in describing the injury caused by the bulb mite on hyacinths states "It is an accepted fact that otherwise

healthy bulbs infested by this species are subject to rot. As the disease develops and the bulb becomes soft, the hypopus stage of the mite appears, and migration to healthy bulbs takes place by means of some of its well known carriers. These healthy bulbs in turn become soft and rot."

Garman (1937) has studied the life history and activity of the bulb mite and reported experiments in which rotten bulbs containing mites were placed in pots of soil just below healthy bulbs. The mites readily migrated from the rotten bulbs and infested the healthy ones, causing them to decay (Fig. 182). Welsford

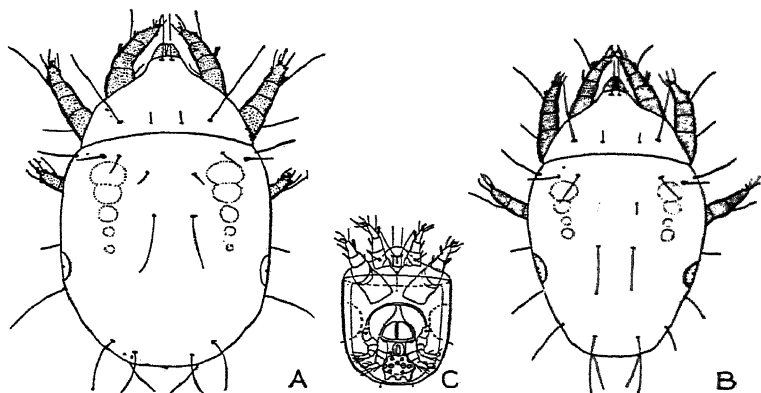


FIG. 181.—The bulb mite. A, adult female, dorsal view; B, adult male, dorsal view; C, the hypopus, ventral view showing the ten suctorial organs by means of which the mite attaches itself to insects to be transported to new localities. Approx. 80 \times . (After Garman.)

(1917), who investigated a bulb rot of narcissus in England, concluded that the nematode [*Tylenchus (Anguillulina) dipsacii*] was more important as a vector than the bulb mite. The micro-organisms responsible for the decay of narcissus bulbs have never been studied intensively, and it is probable that more than one disease is involved.

The narcissus fly (*Eumerus tuberculatus*) and other insects play an important part in the distribution of the bulb mites which, in the hypopial stage, attach themselves to various parts of the insects' bodies and are distributed by the flies as they travel from bulb to bulb (Fig. 183). The mites are distributed also by many other insects that infest decaying bulbs under conditions

that would be conducive to the dissemination of bacteria or spores of pathogenic fungi.

According to Taubenhaus (1913), black rot of sweet potato, caused by *ceratostomella fimbriata* (E. and H.) Sacc., is transmitted from root to root in the storage cellar by mites and red spiders (specific identity not given). It was stated that when these arthropods were transferred from diseased to healthy roots the disease always followed. A pure culture of the pathogen was

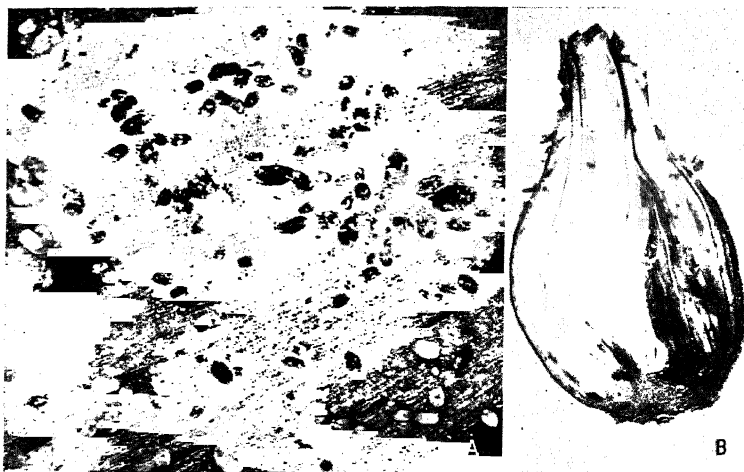


FIG. 182.—A, mites feeding on a bulb scale; B, a hyacinth bulb infested with mites and beginning to decay as a result of inoculation of the tissues by the feeding mites. (After Garman.)

isolated when the mites were taken from diseased roots and allowed to crawl over a poured plate of culture medium.

Mites of various kinds are commonly associated with decaying plants where they are generally referred to as scavengers. This preconceived idea of the nature of their activity together with the known fact that they are wingless has tended to discount their possible significance as vectors of plant diseases. Certain dipterous insects, long known to be associated with decaying plants and generally considered to be scavengers only, have been shown to be important vectors of the microorganisms causing the decay (Leach 1926, 1927), and it is not improbable that some of the mites now classed as scavengers may prove to

be active vectors of pathogenic microorganisms. Moreover, some of these mites are tyroglyphids and are disseminated by flies and other winged insects that feed on decaying tissues. Therefore, the fact that the mites are wingless does not preclude the possibility of their action as vectors. There is need for more thorough study of the significance of mites as vectors of microorganisms that cause rotting of plant tissues.



FIG. 183.—Two flies (*Scatopse pulicaria* Loew) that breed in decayed bulbs, with hypopi of the bulb mite clinging to them. Approx. 11 X. The flies transport the mites to healthy bulbs and the mites in turn transport the fungi that are responsible for the decay. (After Garman.)

Mites as Vectors of "Reversion" in Black Currants.—Reversion, a destructive virus disease of black currants, prevalent in England and in Western Europe, is said to be transmitted by a mite [*Eriophyes ribis* (Westw.) Nal.] (Amos and Hatton, 1927, 1928). This mite (Fig. 173) is also the cause of the condition on currants known as "big bud" in which the buds become much enlarged but fail to open and eventually die. The mites then migrate to new buds and start new infestations. Big bud is not a symptom of reversion but is a distinct manifestation of mite infestation. Reversion is characterized by leaf abnormalities, dwarfing, and sterility. It can be transmitted by grafting but is not transmitted by artificial sap inoculation or

by the seed. For a somewhat more complete discussion of this disease and its vector, see Chap. IX.

NEMATODES

Nematodes, or roundworms, belong to the phylum Nemathelminthes and include many species parasitic on plants or animals

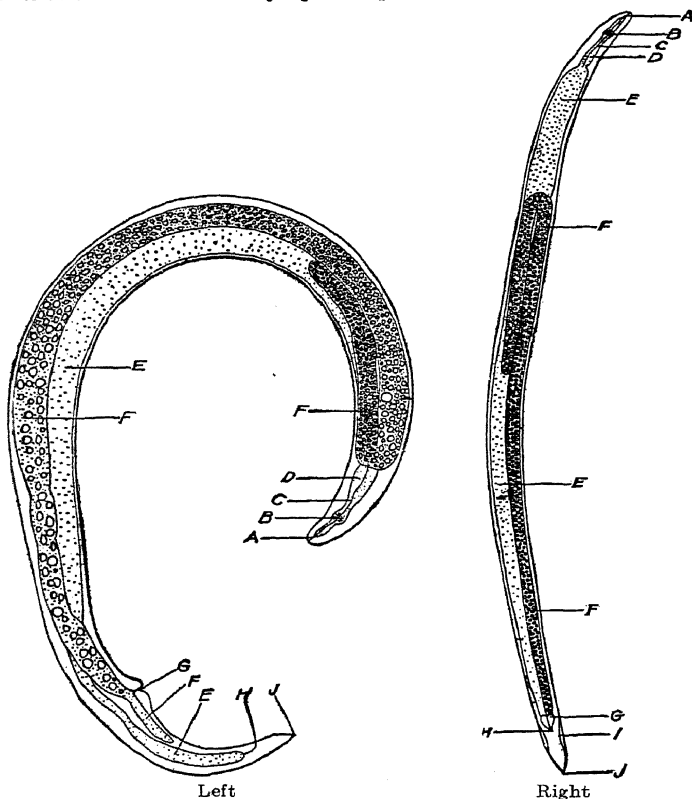


FIG. 184.—*Tylenchus tritici*, a nematode causing a gall of wheat. Left, lateral view of a young female; right, a ventral view of a young male; A, spear; B, anterior esophageal bulb; C, esophageal canal; D, posterior esophageal bulb; E, digestive system; F, reproductive system; G, spicula in male and vulva in female; H, anus; I, bursa of male; J, tail. (After Byars.)

as well as free-living saprophytes. Some species that affect animals are relatively large, often attaining a length of several,

feet; but those affecting plants are small, usually not more than a few millimeters in length. These are often referred to as "eelworms." The anatomy of nematodes is relatively simple (Fig. 184). They have elongated, spindle-shaped bodies. The mouth, provided with hook or stylet in the buccal cavity, leads into a muscular esophagus which serves as a suctorial pump. The intestinal tract is usually a straight tube. The body wall is muscular and is not segmented but is often transversely marked with rings of stiff cuticle. The nematodes move by a characteristic undulatory movement.

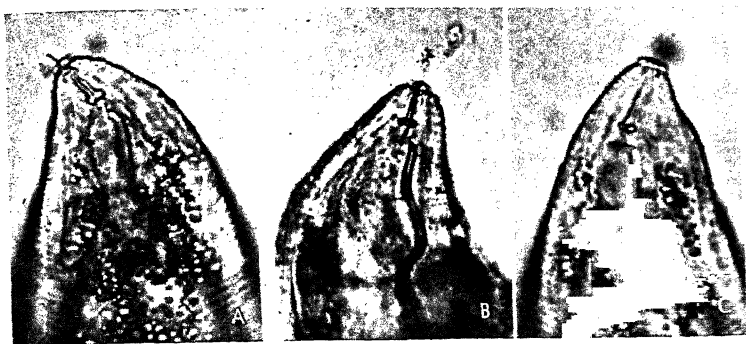


FIG. 185.—Photographs of the anterior end of the root-knot nematode, *Heterodera marioni* showing the action of the mouthparts. A, showing the stylet protruding into the media; B, showing the extrusion of saliva; C, showing the mouth parts with the stylet fully retracted. (After Linford.)

A nematode of the common plant-infesting species is equipped with a minute, hollow axial stylet, or spear, by means of which it pierces the cell walls of infested plants (Fig. 185). As a rule, the nematodes work their way into the tissues through the intercellular spaces. Usually they remain in the intercellular spaces and feed by piercing the cell walls with their stylets and sucking out the cell fluids. Digestion of the cell contents is aided by the injection of saliva through the hollow stylet. The method of feeding of the root-knot nematode (*Heterodera marioni*) has been described clearly by Linford (1937). For a more detailed description of nematodes parasitic on plants and discussion of the diseases caused directly by them, the reader is referred to the treatise by Goodey (1933).

Nematodes and the *Dilophospora* Disease of Cereals.—This is an association of two distinct diseases of cereals, a seed gall caused by a nematode [*Tylenchus tritici* (Steinbuch) Bastian] and a leaf-spotting disease caused by a fungus [*Dilophospora alopecuri* (Fr.) Fr.]. In Europe, the two diseases are usually associated. The nematode galls may occur independently of the fungus disease, but the fungus disease is rarely found on plants not affected with nematodes. The association of the two pathogens has been investigated extensively by Atanasoff (1925) who speaks of it as "coparasitism." He concludes that the fungus disease is dependent upon the nematodes for its transmission and development but that it has a detrimental effect on the nematodes and the disease caused by them.

The nematode (*T. tritici*) (Fig. 184) infects the developing ovaries and causes characteristic globular galls that replace the normal seeds of the affected plants. In each gall are found 10,000 to 15,000 dormant nematode larvae. The galls fall on the ground at harvest or may be planted later with the seed. As soon as the galls come into contact with the moisture of the soil, the nematode larvae become active, leave the gall, and wander through the soil to the nearest host plant. They instinctively find their way into the space between the leaf sheaths of the young cereal plants where they live as ectoparasites. Gradually they work farther and farther between the leaves toward the center until they finally reach the growing point. As the plant grows and the internodes elongate, the nematodes are carried passively upward by the elongating stem. As soon as the head is differentiated, the immature nematodes bore into the young ovaries where they produce sexually, giving rise to thousands of young larvae in each gall. As the plant matures and the tissues become dry, the galls harden and the larvae become dormant, thus completing the life cycle.

The fungus (*D. alopecuri*) causes numerous dark spots, blotches, and stripes on the upper leaves and on other parts of the plant. Spores are formed in great numbers in pycnidia localized in the spots. In the early stages of infection, the fungus grows in between the folded leaves, binding them together so that the developing head cannot escape and is badly deformed and distorted. The nematode galls also become infected with the

fungus, Atanasoff being unable to obtain any galls completely free of *Dilophospora*.

The spores of *Dilophospora* are cylindrical with tufts of bristlelike appendages at one or both ends (Fig. 186). They are approximately 12 microns in length, exclusive of appendages, and 2.2 microns in width. The appendages are 5 to 10 microns

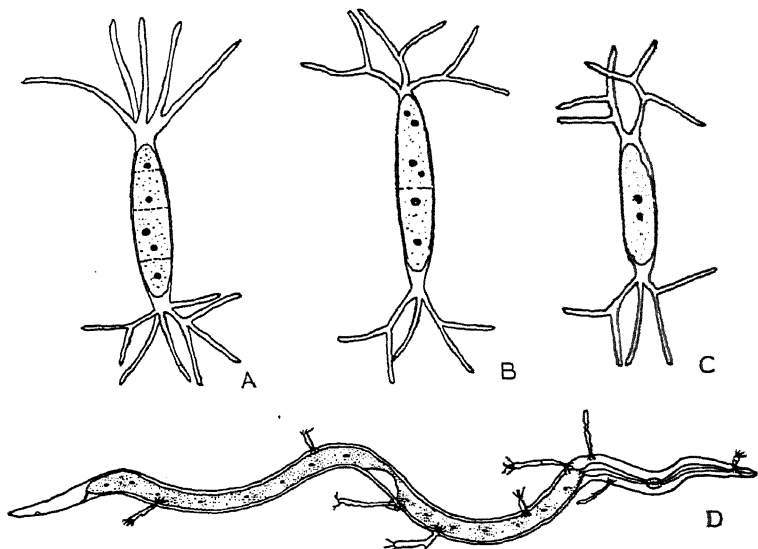


FIG. 186.—*Dilophospora alopecuri*. A, B, and C, spores showing the setae by which they adhere to the nematodes; D, a nematode with spores of *Dilophospora alopecuri* adhering to it illustrating the method by which the spores are transported in between the unfolded leaves to the growing point where they germinate and infect the very young leaves. (Redrawn after Atanasoff.)

in length and about 0.5 micron in diameter at the base. They are either simple or branched.

When the nematodes in the galls become active, the fungus also begins to grow and to form spores which become attached to the body of the nematode larvae by means of the bristles as shown in Fig. 186d. In this way, the spores are transported by the nematode larvae into the intersheath spaces and to the young growing point. Here some of them germinate and infect the very young leaves. The symptoms, however, do not

become noticeable until the leaves have unfolded. Nematodes taken from near the growing point of wheat plants always carry numerous spores attached to their bodies.

When spores were artificially introduced into the plant near the growing point in the absence of nematodes, the disease was not produced. Atanasoff explains his inability to produce the disease by artificial inoculation in the following way: That which was a growing point a few days or even hours ago is now a leaf. The spores introduced artificially in the absence of nematodes are soon pushed upward and outside onto older tissue where infection does not take place. The nematodes avoid this fate by continuously deserting the newly differentiated leaves and moving constantly downward to the undifferentiated tissues of the growing plant.

The nematodes not only bring the *Dilophospora* spores into the central cavity of the plant and carry them subsequently back to the growing point of the plant from its growing out leaves, but the nematodes, sucking or otherwise injuring or affecting the plant cells of the tender and minute leaves, open the way for the penetration of the fungus into the cells and tissues of the leaves and afterwards of the young head.

Additional support of this explanation is furnished by the fact that plants already infected with *Dilophospora* will completely recover and produce normal leaves as soon as the nematodes die, as they sometimes do. It is also noteworthy that the lower and older leaves are never infected by the fungus.

Schaffnit and Wieben (1928) have questioned the importance of the nematodes in the development of this disease. By inoculating the very young tissues of germinating seed with a suspension of *Dilophospora* spores, it was shown that the fungus could infect wheat and rye plants in the absence of nematodes. The conditions that resulted in artificial infection were, however, not entirely typical of the conditions that influence the development of the disease as it occurs in nature.

It is apparent that we are dealing here with a fungus disease of cereals that is transmitted by a nematode, itself a cause of disease on the same plant. Although the fungus may infect its host plant and survive in the absence of the nematode, it apparently is more effective as a pathogen when associated with the nematode that transports the spores to the delicate

tissues of the growing point. The spores are ensured of transportation by virtue of the bristlelike appendages that adhere readily to the mucous covering of the nematodes. In so far as is known, the nematodes derive no benefits from the association.

Root Nematodes as Agents of Transmission of Plant Pathogens.—There are numerous nematodes that infest the roots of plants. Many of these, such as the root nematodes *Heterodera marioni* and *H. schachtii*, cause destructive diseases independently of pathogenic fungi or bacteria. Others are aided in their destructive activity by pathogenic microorganisms which are introduced into the plant tissues by the nematodes. There are many reports in the literature of observed associations of nematode infestations and pathogenic microorganisms, but very few of these have been studied in detail. The majority of the reports deal mainly with observations unsupported by careful microscopic examination and experimental evidence, although there are some exceptions.

One of the more recent reports is that of Kalinenko (1936), who has shown that in Russia three species of nematodes (*Tylenchus multicauda* Cobb, *T. pratensis* de Man, and *Aphelenchus avenae* Bost) attack the roots of two rubber-bearing plants and inoculate them with *Erwinia carotovora* and several other species of bacteria. The combined actions of the nematodes and bacteria are destructive to the plants.

Careful microscopic examination showed clearly that the bacteria were inoculated into the root tissues by the nematodes. It was demonstrated by cultural studies that the bacteria were transported both internally and externally by the nematodes and that the bacteria, having gained access to the root, caused extensive destruction of the tissues independently of the action of the nematodes.

What is apparently a similar association between nematodes and a fungus has been reported by Triffit (1931) from England, where the nematode *H. schachtii* is often a destructive pest of potatoes causing a condition referred to as "potato sickness." It was observed that plants in certain areas in a field often are more severely injured than plants in other areas which are equally as heavily infested with nematodes. It was concluded that when nematodes were the only factor concerned, very little harm was done, the severe injury being caused by the combined

action of the nematodes and a fungus (*Colletotrichum atramentarium*). No detailed histological or cultural study was made of the association.

Millikan (1938) has studied the eelworm disease of cereals (*H. schachtii* Schmidt) in Australia. He comments that "root-rot fungi occur freely in association with eelworms . . . particularly in wheat and in some instances at least, these undoubtedly have been the major cause of poor growth."

It is only reasonable to expect that when plant roots are invaded by nematodes, the resulting wounds would often serve as entrance points for soil fungi or bacteria which are to some extent pathogenic. In most cases, the injury caused by the relatively inconspicuous fungi or bacteria has been attributed to the activity of the more obvious nematodes. There are numerous examples of nematode injury in which fungi have been observed to be involved to some extent. A few of the better known ones will be mentioned briefly.

The root-knot nematode [*H. marioni* (Cornu) Goodey] has long been recognized as a contributing factor in the development of fusarium wilt of cotton. Wilt is always more severe on soil infested with nematodes than on noninfested soil (Young 1928, 1938; Rosen, 1928), and it has been observed that the most wilt-resistant cotton varieties may become very susceptible in nematode-infested soil. The mechanism by which the resistance is broken down is not known, although it is assumed that the more effective inoculation resulting from nematode attack is the determining factor. Rosen states:

The action of nematodes on roots is largely that of producing localized, hyperplastic overgrowths, consisting mostly of soft, parenchymatous tissue and a reduced amount of cork and wood. It seems reasonable to assume that this production of soft, succulent tissue offers an excellent opportunity for the growth of the cotton-wilt parasite, and this appears to be the explanation for the prevalence of cotton wilt on nematode-infested soil.

Arndt (1935) has reported that damping off of cotton seedlings often follows invasion of the roots by nematodes. He states that the nematodes alone, in the absence of fungi and bacteria, are unable to cause damping off.

The eelworm disease of potatoes caused by *Anguillulina dipsaci* Kühn is often destructive in England and Holland. The tubers

are affected, the injury closely resembling that caused by late blight. According to Goodey (1933), "As a rule a general putrefactive rot sets in and spreads throughout the tubers when these are lifted and clamped; consequently where the disease is found the crop should be disposed of and not stored in clamps." The direct cause of the decay in this case has not been studied extensively.

The same nematode also affects narcissus bulbs, and it is stated (Goodey 1933) that "diseased bulbs are rapidly attacked by many other secondary invading organisms including saprophytic nematodes, bacteria, mites, moulds, and fly larvae, etc., till finally a rotting mass is produced within the outer dry scales." Welsford (1917) has studied a destructive bulb rot of narcissus associated with infestation of this nematode. He concluded that the nematode is of greater significance as a vector of decay than the bulb mite.

The burrowing nematode [*A. similis* (Cobb) Goodey] has been shown by Muir and Henderson (1926) to be associated with a root rot of sugar cane in Hawaii. The nematode attacks the sugar-cane roots near the tips, terminating the growth and causing excessive formation of laterals which are in turn infected. The affected parts become red, then brown, and finally purplish black. A fungus is constantly associated with the nematode attacks and, when cultured, produces the same color on artificial media that is found in the affected roots.

Anguillulina (*Tylenchus*) *pratensis* de Man is a nematode that attacks the roots of many different kinds of plants. It is injurious to cereals in Germany and other regions where it has been studied extensively. According to Goodey (1933), the injury is caused not so much by the direct action of the nematode as by the fungi which attack the roots weakened by the nematode, but no careful study of the associated fungi has been made. Steiner (1927) has described and pictured this nematode in the roots of various plants (Fig. 187). He states that "specimens of *Tylenchus pratensis* undoubtedly move slowly but continually through the root tissues. The path prepared by the larvae aids in the spread of other root diseases through the root tissues, especially fungi. Cases have been observed by the writer where numerous chytrids could be seen following what appeared to be the trail of *T. pratensis*."

It will be seen from the preceding discussion that the relationship between nematodes and plant pathogens is in need of further study. The general problem of nematodes and associated fungi and bacteria has been stated by Goodey (1933) as follows:

Because we sometimes find nematodes associated with unhealthy conditions of plants and are unable to assign the disease to some par-

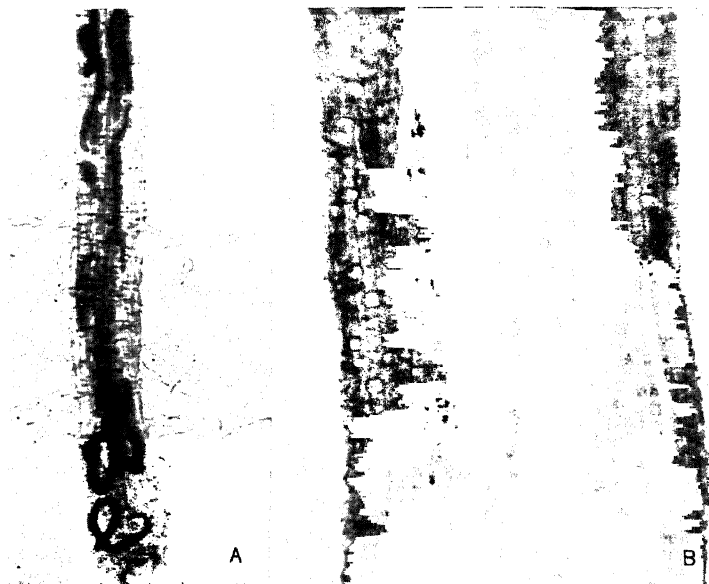


FIG. 187.—Nematodes in roots. A, *Tylenchus pratensis* in a root of a wheat plant (approx. 55 \times); B, *T. pratensis* in a root of lily of the valley (approx. 60 \times). (After Steiner, by courtesy of the U. S. Dept. Agr.)

ticular pathogenic organism, we may be inclined to conclude that the nematodes found are causally related to the diseased conditions. This is scarcely warrantable without a thorough search for other possible sources of disease of a fungal, bacterial, virus, or physiological nature. The whole subject is one calling for much further investigation since at the present time, apart from our knowledge of the obligate parasites, we have but little exact information on the part played by other kinds of nematodes in the microbiology of the soil or in relation to the plant in health or disease.

EARTHWORMS

Earthworms and Clubroot of Crucifers.—Gleisberg (1922) has reported evidence to show that earthworms are concerned in the spread of clubroot of crucifers, a disease caused by a fungus (*Plasmodiophora brassicae* Woronin). Large irregular swellings develop on the roots of affected plants. Spores are formed within the cells of affected tissue and are liberated only after the plant dies and the roots decay. The spores germinate in the soil and form myxamoebae that find their way to the roots of new plants which they infect through the root hairs. Although infested areas of soil do not increase in size rapidly, the rate of spread is often faster than can be explained by the direct migration of the fungus in the soil.

Gleisberg placed 3 to 10 earthworms in pots of infested soil and allowed them to remain for 1 month, then removed them to new pots of disease-free soil which he planted to cabbage. Sixty per cent of the plants were infected in these pots, whereas none were infected in similar pots of noninfested soil to which no earthworms were added. The pathogen, in virulent form, was found to be abundant in the excreta of the worms, and it was concluded that earthworms in nature would carry the spores of *P. brassicae* for some distance in the soil. It was shown that deep plowing to turn under the infested soil is of only temporary value, because the earthworms soon bring enough of the inoculum to the upper layers to reinfest it.

The dissemination of *Plasmodiophora* in this way would be relatively local, because earthworms do not travel for very great distances. There is a possibility that they may be indirectly responsible for long-distance transmission by birds that feed upon earthworms. There is no experimental evidence, however, to show that this ever occurs.

SLUGS

On several occasions, slugs have been reported as agents of dissemination of fungus spores or bacterial plant pathogens, but as a rule they are of relatively little importance as vectors of plant diseases. As early as 1896, Wagner reported experiments with slugs (*Helix hortensis* Müll., *Succinia putris* L., and *Arion subfuscus* Drap.) showing that they effectively dissemi-

nated spores of several species of pathogenic fungi. The spores were disseminated both externally in the slime and internally in the fecal excretions. The spores of all species of fungus tested passed through the intestinal tract of the slug uninjured, and such spores were virulent when tested by artificial inoculation. In a number of experiments, slugs were starved for several days, then allowed to feed, first on diseased plants, then later on healthy plants to which the diseases were transmitted [*Plasmopera nivea* (Unger) Schroeter on *Aegopodium podagraria* L., *Bremia lactucae* Regel on *Sonchus oleraceae* L., and *Peronospora parasitica* (Pers.) on *Dentaria bulbifera* L. and others]. In no case, however, was it shown that the transmission by slugs was of major importance in the spread of the disease in nature.

Smith (1897) has reported successful transmission of black rot of cabbage [*Phytophthora campestri* (Pammel) Bergey et al.] by slugs, but this method of transmission is of little significance in nature. Buller (1909) has reported that many slugs feed on the fruiting bodies of the fleshy fungi, ingesting large quantities of spores. He reviews the work of Voglino who found germinating spores of several species of the Hymenomycetes in the intestinal tracts of slugs. Toads that feed on slugs also harbor viable fungus spores in their intestines. Voglino believed that the slugs played an important role in the propagation of the fungi by providing suitable conditions for spore germination. Buller, on the other hand, more logically concludes that "although it may be true that slugs help in the local dispersal of spores in a wood or field and provide conditions for their germination, these animals, owing to their slow rate of movement, could scarcely act as agents in spreading fungus species from wood to wood when they are separated by considerable distances."

Gravatt and Marshall (1917), in an experimental greenhouse test, observed slugs eating the teliospores of *Cronartium ribicola* and demonstrated that some of them were viable after passage through the digestive tracts of the slugs, but there was a marked decrease in the percentage of spores that germinated. It was concluded that these animals were of relatively little practical importance as vectors of the disease.

Similar observations on the dissemination of cereal-rust spores by the imported garden slug (*Agriolimax agrestis*) were reported

by Granovsky and Levine (1932). Slugs that had been allowed to feed on rust pustules in the greenhouse were placed on healthy seedlings. Rust infection invariably appeared along the trails of the slugs as marked by the mucous secretion. Infection was obtained also by using the excreta and intestinal contents of slugs as inoculum. The spores were apparently uninjured by passage through the slugs, but the latent period of the rust was somewhat prolonged. Transmission of rust by slugs in the greenhouse might be of significance from the standpoint of experimental work but would be of little significance in the epiphytology of cereal rusts in nature.

BIRDS

From time to time, various workers have expressed the opinion that birds are important vectors of certain plant diseases. However, there has been relatively little careful study of the question. The most complete investigation of the subject on record is the study of Heald and Studhalter (1914), dealing with birds as vectors of the chestnut-blight fungus. These workers made a study of the spores carried on the bills, feet, and feathers of 36 birds belonging to nine different species. All the birds examined were shot on infested chestnut trees. The species represented in the study are as follows: (1) the hairy woodpecker (*Dryobates villosus*), (2) the downy woodpecker (*D. pubescens medianus*), (3) the junco (*Junco hyemalis*), (4) the flicker (*Colaptes aureatus luteus*), (5) the white-breasted nuthatch (*Sitta carolinensis*), (6) the brown creeper (*Certhia familiaris americana*), (7) the golden-crowned kinglet (*Regulus satrapa*), (8) the sapsucker (*Sphyrapicus varius*), and (9) the black-and-white creeper (*Mniotilta varia*). Of these, the hairy woodpecker and the flicker were the only ones not found carrying spores of *Endothia parasitica*, the blight pathogen. Of the 36 birds examined, 19 were found to be carrying spores of the fungus. The estimated numbers of viable spores carried by two downy woodpeckers were 757,074 and 624,341, respectively; and a brown creeper carried 254,019 viable spores.

The old cankers of chestnut blight are frequently infested with bark-boring insects as pointed out by Craighead (1916), and the birds often riddle the cankers in search of the insects. Such a riddled canker of chestnut blight is shown in Fig. 188. In this

way, the birds become thoroughly contaminated with spores of the fungus.

The birds, killed 2 to 4 days after a period of rainy weather, always carried more spores than those killed in dry weather. The spores carried were all pycnidiospores; ascospores were never found on the birds. The authors conclude that birds are important vectors of the fungus but that because of the predominance of wind dissemination of ascospores they are not of great significance in local spread. They believe, however, that they are responsible for the so-called "spot" infections at some distance from areas of general infection.



FIG. 188.—A chestnut canker that has been riddled by birds while feeding on insects under the bark. Birds became contaminated with the spores in this way and aided in the dissemination of spores over long distances. (After Heald.)

Inasmuch as the birds involved in the study are migratory, they are potentially important vectors for long-distance spread of the fungus. It is entirely possible that by starting new centers of infection they contributed materially to the rapid spread of chestnut blight throughout the range of the chestnut forests in this country.

The extent to which these birds actually transmit the disease depends largely upon the opportunity they have for inoculating healthy trees. Heald and Studhalter (1914) state that "each time the bird climbs or creeps over the trunk or limbs of a tree some of the spores may be brushed off and may lodge in crevices or on the rough bark. From this position they may be washed down into wounds by rain and may thus cause infections." A still more effective method of inoculation not mentioned by

these authors is provided by the habit many of these birds have of feeding on the cambium of healthy trees. Nearly all the woodpeckers and sapsuckers that feed on bark-boring insects also include fresh cambium in their diet (Roberts 1932). The wounds made in the bark of healthy trees would be ideal infection courts; and if the bird had fed recently on insects from a spore-laden canker, infection would follow almost certainly.

Birds (woodpeckers and sapsuckers) that feed on the elm bark beetles in elm trees infected with the Dutch elm disease have been suspected of transmitting the disease, but no careful study of the subject has been reported. In view of the migratory nature of these birds and their habits of feeding on fresh cambium as well as on the insects from infected trees, it is very likely that they may be responsible for starting centers of infection at some distance from the known area of general infection. Transmission by bird vectors may offer a possible explanation of the few reported cases of trees infected with the Dutch elm disease in the absence of beetle infestation.

Birds as Vectors of Mistletoe.—The mistletoes cause well-recognized diseases of forest trees. They rarely, if ever, kill a tree; but they often do cause severe malformation, sap the vitality of the tree, and greatly reduce its rate of growth. The mistletoes are semiparasites which derive part of their nourishment from the sap stream of living trees. The xylem vessels are tapped by rootlike haustoria through which they withdraw water and mineral salts in solution. They are not dependent upon the tree for carbohydrates, because they possess chlorophyll and are able to synthesize their starch.

The mistletoes are seed-bearing plants (Fig. 189) and are transmitted from tree to tree entirely by seeds which are disseminated largely by birds. The role of birds in disseminating mistletoe has been described by Bray (1910). The seed of the mistletoe, when ripe, is enclosed in a clear, sticky pulp covered by a semitransparent skin. The sticky pulp is clearly an adaptation for seed dispersal. The seed and its covering, constituting the berry, are utilized as food by several species of bird. The mockingbird, the cedar waxwing, and the robin are among the most important birds that feed upon the berries, disseminating the seeds. The birds ingest the seeds and may either pass them out in excrement or regurgitate them. The seeds also adhere

readily to the feet or to the beak, later to be wiped off on a branch. The seeds by virtue of their sticky coating adhere to the branches and in moist weather germinate and infect the tree. The seeds are obviously poorly adapted to wind dissemination.

Bray has observed that the point of first infection on a tree often is determined by the perching habits of the birds that feed upon the berries. Large areas were noted in which there was a predominance of cases where a single plant of mistletoe



FIG. 189.—A female plant of western dwarf mistletoe, with mature berries on ponderosa pine. The sticky pulp of the berry causes the seed to adhere to tree branches, where they germinate. They also adhere to the feet and beaks of birds and may be transported long distances by them. (After Boyce.)

grew from a point near the apex of the topmost branch, a choice perching place of the mockingbird. This point of primary infection is ideal for further infection of the lower branches by falling seeds.

The dwarf mistletoes, which cause destructive diseases of conifers in Western North America, also produce their seed in a sticky matrix. On maturity, the berries develop a strong internal pressure which slowly increases until a slight disturbance causes them to explode, forcibly ejecting the seed for a distance of several feet. The sticky coating causes them to adhere to the surrounding branches on which they may fall. Local dissemination is accomplished in this way, but for dispersal over longer

distances dependence must be placed upon birds. The birds transport these seeds in the same ways that they transport the seeds of the true mistletoes.

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CHAPTER XII

THE ANATOMY AND PHYSIOLOGY OF PLANTS IN RELATION TO INFECTION AND INSECT VECTORS

In the process of evolution, plants have developed a number of natural defenses against unfavorable environmental factors, insect pests, and infection by microorganisms. At the same time, the pathogenic microorganisms and insect pests have evolved means of overcoming to some extent these protective adaptations. The end result has been a certain degree of equilibrium in the struggle, involving the interaction of plant, pathogen, and insect. In order to have an adequate concept of the role of insects in the development of plant diseases, it is necessary to consider in some detail the natural defenses of plants, as well as the ways in which the pathogens or insects overcome or circumvent them. Only in this way can we properly evaluate the part played by insects in the processes of infection.

The plant should be visualized as a living organism composed of a number of delicate tissues that must be protected against the vicissitudes of both the living and nonliving environment. Each tissue and each cell of the plant have a number of functions to perform, and their nature is usually determined by their functions. Since protection is only one of the many functions of plant tissues, compromises have been necessary so that perfect protection has never been achieved. Chief among the living components of the environment, against which protection is necessary are insects and pathogenic microorganisms. In this chapter, certain tissues and physiological processes will be considered from the viewpoint of their influence upon the susceptibility of plants to the attack of microorganisms or insects, or both combined.

The Epidermis.—In the early stages of growth, the entire outer surface of any higher plant is covered with a single layer of specialized cells, the epidermis (Fig. 190). The epidermis forms a complete covering except at a few points such as the stomatal

openings, hydathodes, and nectary glands and in the growing points where the cells are still meristematic, the epidermis not yet having been differentiated. The epidermis is made up of living cells, but all except the guard cells of the stomata are devoid of chlorophyll. With the exception of such specialized cells as root hairs and stomatal cells, the structure of the epidermal cells is designed primarily for protection. The outer and radial walls of

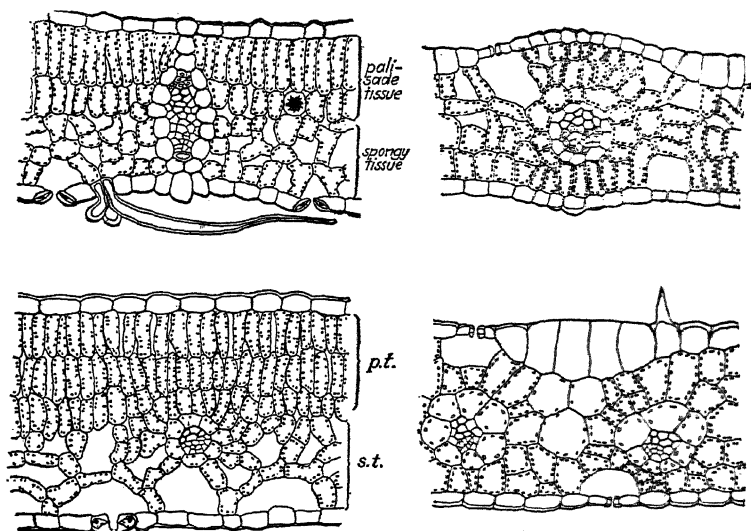


FIG. 190.—Cross sections of leaves showing the epidermis and some of its protective features. Note the thickened outer wall and the arch or domelike curvature it forms. (After *Eames and MacDaniels.*)

epidermal cells are often thickened to give strength and to resist mechanical injuries. The outer wall is curved to form a domelike structure that gives added resistance to pressure from without. Almost the entire surface of the epidermis, on the aerial parts of the plant, is covered with a layer of a waxy substance known as "cutin." This is called the "cuticle" and constitutes a very effective barrier against infection by many microorganisms (Fig. 191). The cuticle is highly impervious to water and air and is resistant to chemical action. No enzyme is known that will dissolve it.

A number of phytopathogenic fungi are able to penetrate the cuticle by mechanical means, others enter the plant through natural openings, but many depend entirely upon wounds. Those which penetrate by mechanical means utilize the force of

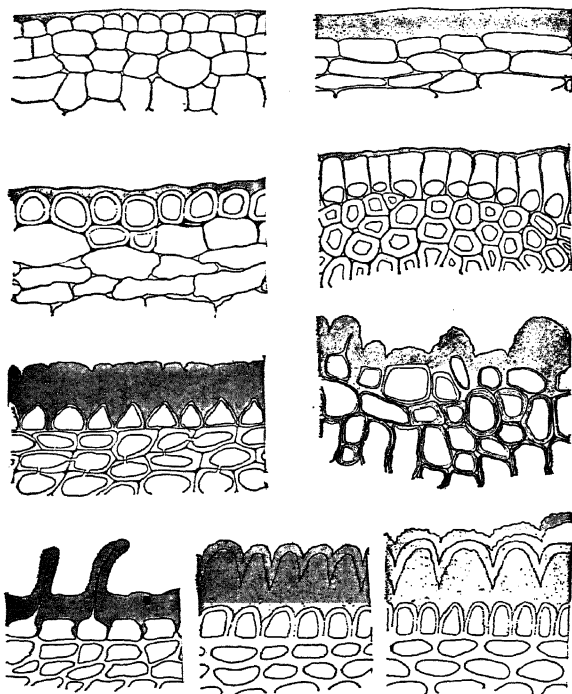


FIG. 191.—Sections of the epidermis and adjacent tissues of several different plants showing the cuticle (shaded areas). The cuticle is composed of cutin, a substance highly resistant to chemical action. Fungi are unable to produce enzymes that will dissolve it. (After Eames and MacDaniels.)

growth pressure. The germinating spore forms a thick-walled spherical body closely appressed to the cuticle (Fig. 192). The entire protoplasm of the germinating spore is condensed into this "appressorium." Growth is renewed at the point of contact of appressorium and cuticle, and a very thin hypha is pushed through the cuticle. After the cuticle is penetrated, the hypha

enlarges to normal size and continues to grow. This method of penetration is common among certain groups of fungi, but the majority of pathogenic fungi gain entrance in some other way.

Wounds serve as the most important infection point for plant-pathogenic bacteria. No bacteria are known that are able to penetrate the plant through unbroken cuticle. With the exception of stomata, lenticels, hydathodes, the floral organs, and the noncutinized root hairs, wounds are the only avenue of entrance for the bacterial pathogens.

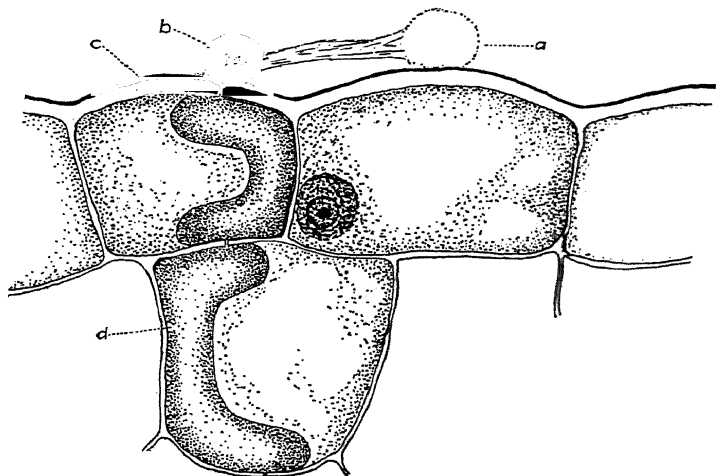


FIG. 192.—A semidiagrammatic drawing showing the method of cuticle penetration by *Colletotrichum lindemuthianum* the pathogen of bean anthracnose. a, Empty spore after germination; b, appressorium; c, cuticle of bean plants; d, fungus mycelium in host cell.

The cuticle is often covered with minute particles of wax, or "bloom," which, because of its high surface tension, very effectively sheds water, preventing it from wetting the surface of the plant. Plants or plant parts that shed water readily usually escape direct infection with greater frequency than plants that wet more easily. Stomatal penetration by bacteria is also rare on plants with a heavy bloom. This fact can be attributed to the absence of a film of water connecting the surface of the epidermis with the substomatal chamber. Such a film appears to be necessary for stomatal infection by bacteria.

Although the cuticle may serve as a barrier against pathogenic microorganisms, it is not always effective against the feeding and oviposition of many insects. A thick or tough cuticle, however, might easily discourage certain species of insects and cause them to show a preference for the varieties or species with a thinner

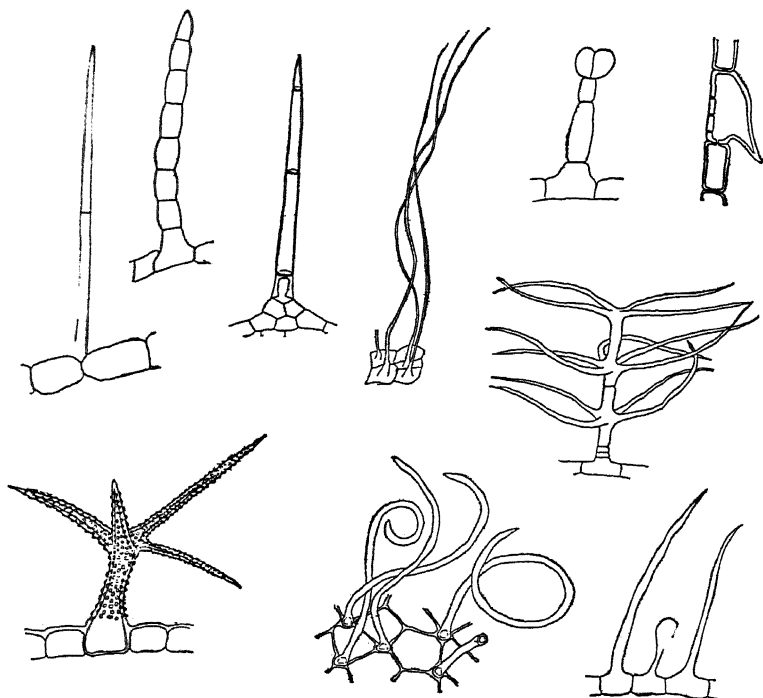


FIG. 193.—Trichomes or plant hairs from several different kinds of plants. These are all modified epidermal cells. They serve many different functions, one of which is protection against insect and fungus enemies. (After Eames and MacDaniels.)

cuticle. In such cases, if the insect were a vector of a disease, the nature of the cuticle would be an important factor in the ability of the variety to escape infection. Susceptible varieties that escape infection in this way are said to be "klendusic." Numerous examples of klendusic varieties of crop plant have been reported, but for many of them no satisfactory explanation of the

disease-escaping qualities is known. A study of the nature of the cuticle of such plants and its relation to the feeding and oviposition of insect vectors should be made.

On many plants, certain epidermal cells are often modified in structure to form hairlike projections commonly known as "trichomes" (Fig. 193) which vary widely in form and may serve different functions. A plant with numerous trichomes is said to be "pubescent"; one with few or none is "glabrous." The degree of pubescence of a variety often determines the extent to which certain insects will feed or breed on it. When the size or abundance of the trichomes is sufficient to interfere greatly with the insects, they will avoid the pubescent varieties and seek the glabrous ones. Consequently, the pubescent varieties often escape diseases caused or transmitted by the insects. Perhaps the most striking examples are afforded by the pubescent varieties of clover reported by Hollowell, Monteith, and Flint (1927) and Monteith and Hollowell (1929) and the pubescent varieties of soybeans described by Johnson and Hollowell (1935). The pubescent varieties of these crops suffer much less from hopperburn, because the leaf hoppers [*Empoasca fabae* (Harris)], responsible for the burn, prefer the more glabrous varieties and feed and breed on them to the exclusion of the pubescent ones (Figs. 55 and 56). A knowledge of this anatomical relationship should be of considerable value in breeding for superior disease-escaping varieties of these crops.

A similar relationship between apple varieties and leaf-hopper injury has been reported by Schoene and Underhill (1937). These authors showed that "the susceptibility of apple varieties to leaf-hopper injury varied inversely with the amount of pubescence of the foliage." The recognition of the correlation of such visible characters with disease-escaping qualities should be of value in apple breeding as well as in the selection of varieties for planting in regions where leaf-hopper injury is prevalent.

The Stomata.—The openings in the epidermis, through which interchange of gases between the atmosphere and subepidermal cells occurs, are called "stomata" (Fig. 194). These usually are well distributed over the surface of leaves and on the surface of green stems. The stomata are necessary openings in the protective epidermis, but they are utilized also as the chief avenue of entrance by many disease-producing fungi and bacteria. Many

pathogens, however, cannot infect in this way, even though the inoculum may enter the stomata and reach the intercellular spaces. Such pathogens usually require wounds for successful infection. Where stomatal infection is the usual method, insects

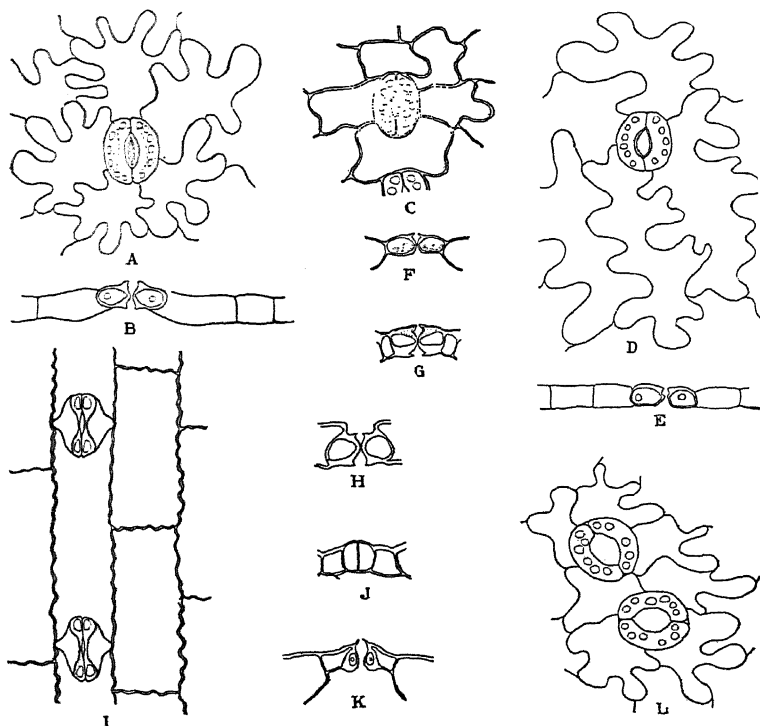


FIG. 194.—Stomata of several different plants, A, C, D, I, and L in surface view. B, C, F, G, H, J, and K in cross section. Stomata are important avenues of entrance for both fungi and bacteria. They are of no significance in insect feeding. (After Eames and MacDaniels.)

are of little importance in ingression, although they may be important in disseminating the inoculum.

The stomatal openings are so small that they have little or no influence on the feeding of insects. Some are sufficiently large to permit the insertion of the mouth parts of the smaller sucking insects, such as aphids, but as a rule these insects show no prefer-

ence for the stomata. Their mouth parts are capable of penetrating the cuticle without difficulty and they usually follow the intercellular spaces of the underlying tissues.

Stomata rarely serve as avenues of infection for the viruses, although Duggar and Johnson (1933) obtained a small amount of infection by spraying uninjured tobacco leaves with a suspension

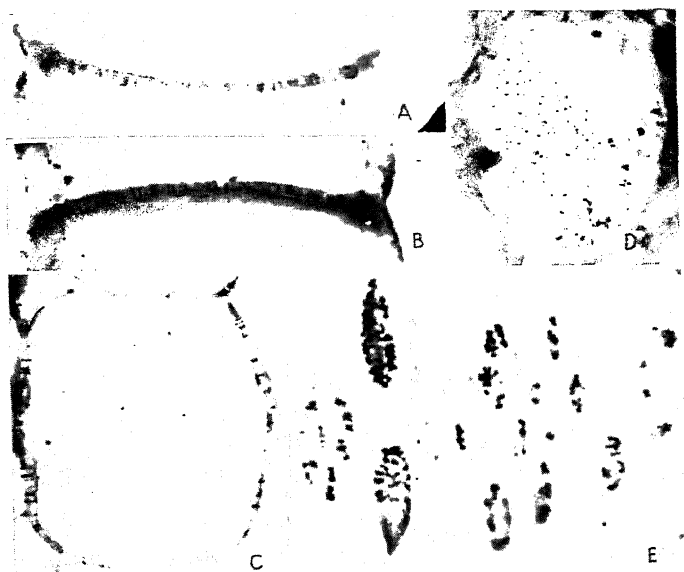


FIG. 195.—Plasmodesmata. Photomicrographs of cell walls of the tobacco plant stained to show the strands of protoplasm which connect the protoplasts of adjacent cells. *A*, *B*, and *C*, seen in cross section; *D* and *E*, surface view of the cell walls; *A* and *B*, 880 \times ; *C*, 260 \times ; *D*, 440 \times ; and *E*, 1,020 \times . (After Livingston.)

of the mosaic virus. Since the virus particles must come in contact with living protoplasm in order to infect, infection through uninjured tissues could be explained only by assuming that some of the virus particles reached the plasmodesmata.

Plasmodesmata.—In nearly all plant tissues, the protoplasts of the individual cells are connected with each other by fine strands of cytoplasm that pass through minute openings in the cell walls. These protoplasmic bridges are known as “plasmodesmata” (Fig. 195). There is considerable evidence to show

that certain viruses may spread from cell to cell through these structures. Drake *et al.* (1934) have observed that aphids and other similar insects often introduce their setae into the walls between the cells and feed without puncturing the cells. It is suggested by these authors that the plasmodesmata serve as important structures in the infection of plants by viruses through the agency of aphids and similar insects.

The Hydathodes.—Under conditions of high humidity and low transpiration coupled with high soil moisture, plants often exude

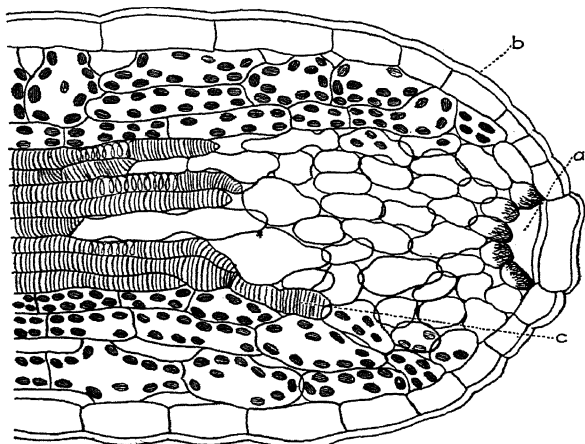


FIG. 196.—A diagrammatic drawing of a section through a hydathode, or water pore, of a leaf. Note that the water pore is located near the end of a vein. Under conditions of high humidity, water from the vessels may pass through the intercellular spaces and be exuded in drops through the pore. *a*, pore; *b*, epidermis; *c*, terminal cell of a xylem vessel.

water from the tips and margins of the leaves. The openings through which the water is exuded are called “hydathodes” or “water pores” (Fig. 196). They are located near the ends of the veins and are directly connected with the water-conducting vessels. The loss of water through the hydathodes is called “guttation,” and the drop of water that is formed on the leaf margin over the hydathode is spoken of as a “guttation drop” (Fig. 197). Water of guttation is relatively pure although it usually contains a small amount of nutrient materials. Some plant pathogens, especially bacteria, may enter the plant through

the hydathodes. They first enter the guttation drop, where they may find nourishment sufficient to support growth and multiplication. As transpiration increases, some of the guttation water may be drawn back into the leaf, and the bacteria are drawn in with it. It has been observed that insects, especially flies, frequently visit and feed upon the guttation drops and in all probability introduce bacteria into them. This is said to be an important method of infection of cabbage by *Phytophthora campestri*, the black-rot pathogen (Smith 1911). Hildebrand



FIG. 197.—Cabbage seedlings growing in a humid atmosphere and bearing drops of guttation water.

(1937a) has shown also that fire-blight bacteria may infect through the hydathodes of the sepals of apple blossoms.

The guttation drops have been considered as a possible avenue of infection for virus diseases, but Caldwell (1930, 1931) has shown that a virus does not infect when added to the guttation drop unless the adjacent plant cells are injured. The virus particles appear unable to diffuse through an uninjured protoplasmic membrane. In a like manner, the guttation drops on a plant infected with a virus disease do not contain the virus unless the tissues have been wounded. It would seem, therefore, that hydathodes are probably of little significance as avenues of infection for virus diseases.

Nectaries and Other Floral Organs.—Exposed tissues, poorly or not at all covered with a cuticle, are found in the flowers of higher plants. These unprotected tissues are usually susceptible to direct infection by many pathogens. The nectary glands, the anthers, and the stigmatic surfaces are especially favorable places for infection without the aid of wounds. The exposed cells often have no protective cuticle and in addition are usually covered with a sugary solution that is very favorable for germination of fungus spores and growth of bacteria. Some nectary glands are covered with an epidermis, the nectar exuding through stomatal openings. In nectaries of this type, the nectar forms a

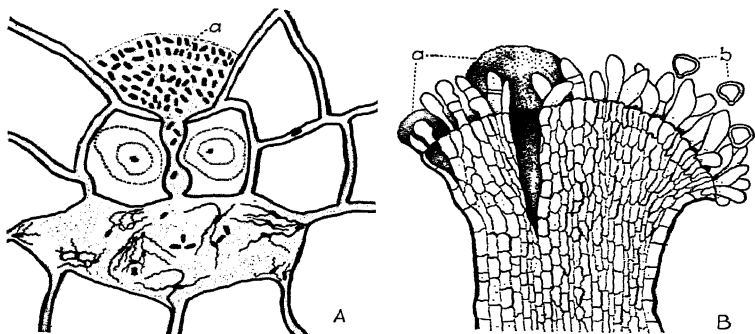


FIG. 198.—Diagrammatic drawings showing methods of blossom infection by the fire-blight bacteria; A, through a nectary stoma; a, bacteria in a drop of nectar. B, through a stigma; a, bacterial mass penetrating the intercellular spaces of the stigmatic surface; b, pollen grains. (After Hildebrand.)

continuous fluid connection from the exuded nectar drops into the intercellular spaces (Fig. 198). Certain species of pathogenic bacteria multiply rapidly in nectar and utilize the nectar glands as avenues of entrance.

Insects of many kinds visit flowers regularly in search of nectar or pollen and in so doing often disseminate inoculum to the very place where infection is most readily accomplished. As examples of this may be mentioned fire (blossom) blight of orchard fruits infecting through nectaries, stigmas, anthers, stomata, and hydathodes and spread by bees, flies, and other insects; the anther smut of pinks infecting through the stigmas and spread by the sphinx moths; the seed blight of clover infecting through the stigmas and spread by bumblebees; and ergot of grasses infecting

through the floral organs and spread by many different kinds of insect. All of these diseases are regularly transmitted by one or more species of insects that habitually visit both healthy and diseased flowers of susceptible plants in search of food, and the unprotected floral parts form the chief and often the sole avenue of entrance for the pathogens.

The Periderm.—In the stems of all plants having secondary growth, the epidermis is eventually replaced by another protective covering called the “periderm.” This is composed largely of cork cells and is even more effective than the epidermis in

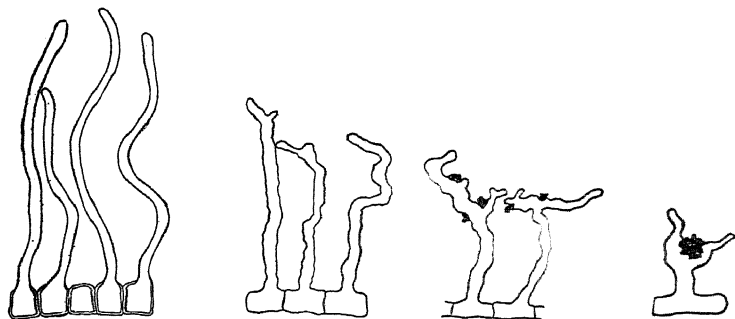


FIG. 199.—Root hairs. These are specialized epidermal cells through which solutions are absorbed from the soil. They are short-lived, persisting for a few days only, after which they die. The adjacent cell walls quickly become suberized, preventing much infection from soil organisms. (After Eames and MacDaniels.)

protecting the plant against infection by microorganisms. Cork cells are nonliving and possess walls that have been infiltrated with suberin, a substance similar in physical and chemical properties to cutin. The suberized cork cells are highly impervious to air and water. The periderm may cover the entire plant except the root tips and the buds, leaves, and flowers. It is the bark of woody plants and the corky covering of all roots. No bacteria and relatively few fungi are capable of penetrating, without aid, the unbroken periderm. However, this protection does not prevail against many insects, such as bark-inhabiting insects which are responsible for the ingress through the periderm of many destructive plant pathogens. *Ceratostomella ips*, the fungus causing blue stain of conifers transmitted by the pine bark beetles, and *C. ulmi*, causing the Dutch elm disease transmitted

by the elm bark beetles, are good examples of pathogens that gain ingress through the bark with the aid of insects.

With the exception of the root tips and the short zone of absorption, covered with root hairs, the entire root system is protected by a periderm of suberized cells. The root periderm is an effec-

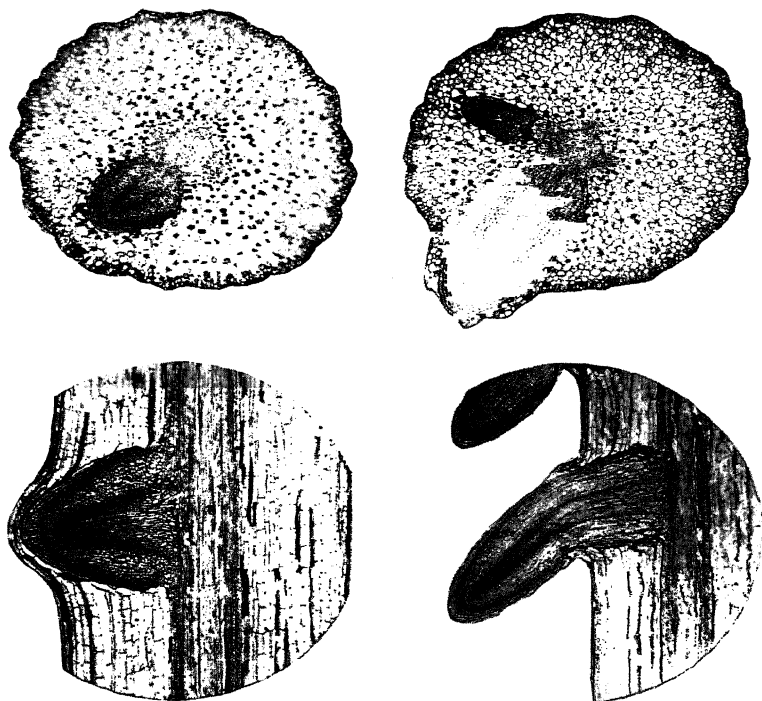


FIG. 200.—Sections of a root of *Salix nigra* showing the endogenous origin of secondary roots and the natural wounds made in the cortex. Such natural wounds in certain plants serve as portals of entry for some soil-borne fungi. (After Eames and MacDaniels.)

tive protection against many of the pathogenic organisms that thrive in the soil. Yet despite this protection many pathogens infect through some underground part of the plant. It is generally assumed that many of them enter through the root hairs (Fig. 199), which are not protected by a cuticle, but in many cases the evidence is lacking. Natural wounds formed by secondary

roots (Fig. 200) have been shown to serve as portals of entry for a few soil-borne fungi, but the extent to which these wounds are significant in root infection is not well known. Because of the obvious difficulty of observing underground infection, the exact place and method of such infection is known for only a few pathogens. Many insect pests of plants are known to spend all or part of their life history underground, and many of them feed on the roots of plants. Where the root feeding is not excessive, it usually escapes observation. Although a few cases of root infection through insect wounds have been well established, the extent to which insect wounds on roots serve as infection courts for plant pathogens is not known. There is room for much study on the soil-inhabiting insects in relation to those diseases which infect through the underground parts of plants. As examples of known root inoculation by insects may be mentioned crown gall, inoculated by white grubs and other soil insects, and the bacterial wilt of corn, inoculated by the southern corn root worm and the seed-corn maggot.

Wound Cork.—Cork tissue formed in response to a wound is known as wound cork. Practically all plants are capable of forming cork tissue in the process of healing a wound. The physiology of wound-cork formation has been extensively studied. A good discussion of the subject is given by Priestly and Woffenden (1922, 1923). When plant tissue is wounded, those cells closest to the surface of the wound die within a few hours; but, in the process, the chemical and physical nature of the cell walls undergo a striking change. The fatty acids in the cell sap are oxidized and are precipitated in and on the surface of the cell walls, making them relatively impervious to water. This is the so-called "blocking" process that prevents the excessive loss of water, and it has been demonstrated that the blocking tissue is an effective barrier against infection by many pathogens. After the blocking process is complete, the cells beneath this tissue become meristematic, forming a cork cambium the cells of which divide to give rise to new layers of cells which are quickly suberized and form a new periderm (Fig. 201). An abundant supply of oxygen and a supply of reserve food materials are necessary for wound-cork formation. The healing of wounds in this way constitutes one of the most effective natural protections of plants against infection. There are certain insects, however, that burrow into

plant tissue and make wounds that are more or less continuous over a relatively long period of time. Such insects, by interfering with the healing process, overcome, to a considerable extent, the protective effect of wound-cork formation. The activity of the seed-corn maggot, in inoculating potato seed pieces with the bacteria that cause blackleg, is of this nature. The larvae of this insect burrow into the seed pieces, inoculate them with the soft-rot bacteria, and destroy the wound tissue as fast as it is formed.

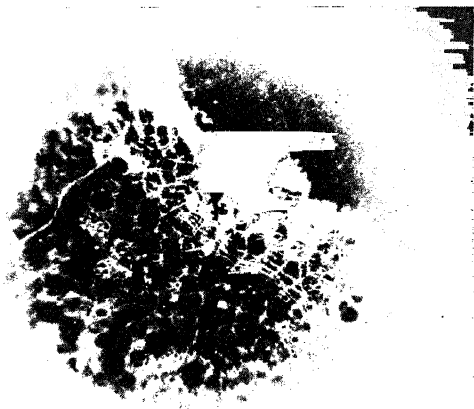


FIG. 201.—A section through a healed wound in a potato tuber. Note the dark irregular-shaped cells that compose the blocking tissue and the layer of more regular brick-shaped cells making up the new wound periderm that has formed from a cork cambium.

The continual wounding of the tissues by the maggots ensures successful infection by the bacteria.

Callus.—When a wound is of considerable size and involves the cambium, another kind of wound tissue known as “callus” is often formed. Callus is composed of soft parenchymatous tissue produced rapidly by the cambium (Fig. 118). The outer cells of the callus tissue differentiate and become suberized to form a protective bark. The continued growth of the callus tissue permits the healing of large wounds, such as those made in pruning when large limbs are cut off. Callus tissue, like wound cork, is a relatively effective barrier against infection by micro-

organisms; but its soft and succulent nature makes it an attractive food for certain insects. The insects, by acting as vectors of plant pathogens, often overcome the protective action of the callus and cause it to function as an infection court. This situation is well illustrated by the relationship of the woolly aphid to the perennial canker of apple trees, in which reinfection occurs each year through wounds made in the callus tissue by the aphids (McLarty 1933 and Childs 1929). Delicate, blisterlike swellings are formed on the callus as a result of the aphid feeding. During the winter the blisters freeze and collapse, providing a suitable point of entrance for the spores of *Gleosporium perennans* which are ever present in the old cankers. Because the trees are dormant when this occurs, infection is accomplished and considerable tissue is destroyed before renewed growth of the callus can take place.

Latex Ducts.—With the discovery of plant-parasitic protozoa (Lafont 1911) which are confined almost entirely to laticiferous plants and are exclusively insect-transmitted, the latex ducts assumed a new significance in the study of plant diseases and their transmission by insects. As pointed out by Holmes (1925c), failure to understand the nature of latex ducts and the relation of the protozoa to them often leads to a misinterpretation of the facts.

The latex ducts are living cells. They contain many nuclei and ramify for long distances through the tissues of the plant. The protoplasm contains large elongate vacuoles which are filled with the milky latex. The ducts have no opening to the outside of the plants, and the protozoa are therefore dependent upon insects for their introduction into the latex cells. The protozoa do not occur in the protoplasm but are found only in the latex of the vacuoles.

There are two different kinds of latex duct, classified according to their mode of origin. One type, usually designated "latex cells," arises by the elongation of a single cell which grows and branches in the manner of a fungus mycelium. Each cell is a distinct unit, and the individual cells do not anastomose with one another.

Another type of latex duct arises from many different cells that unite by dissolution of their adjacent end walls. These ducts are usually referred to as "latex vessels." Latex vessels

are characteristic of such plants as lettuce and other laticiferous composites, whereas the latex cells are found in the euphorbias and milkweeds in which most of the endophytic protozoa are found. The two types of latex duct are represented in diagrammatic form in Fig. 175.

Physiology and Chemical Composition.—The activities of insect vectors are influenced not only by the anatomy of their host plants but also by their physiology. Considerable evidence is available to show that differences in chemical composition of different varieties of plant greatly influence the degree to which they are attacked by certain insects. Many species of plant possess qualities of insect resistance that are obviously based on physiological differences. In many cases, the direct injury caused by the insects may not be sufficiently great to make the differences in resistance obvious or of much economic importance; but if the insect happens to be a vector of a plant pathogen, its preference or dislike of a given variety may be of great significance in the epiphytology of the disease. This is especially true of the virus diseases and their insect vectors, although it applies equally well to all diseases of parasitic origin. Inspection, roguing, and certification of seed or plant parts have become standard procedures in the control of many virus diseases. The rapidity with which a virus spreads in nature often determines the success of certification as a control measure, and the rapidity of spread is often determined by the habits of the vector. Varieties that are preferred by a given insect vector would be more likely to contract the disease than those shunned by the insect, although the varieties may be equally susceptible to the virus when inoculated. Plants that escape infection for this or some similar reason, although they are susceptible to the disease when inoculated, are said to be "klendusic" (Rankin 1927). Because of the significance of klendusity in the practical control of virus disease, this character is deserving of more attention than it has received. In the production of new varieties of crop plants, breeding for klendusity may be of as much practical importance as breeding for true resistance. If the disease is transmitted in nature by insects, breeding for klendusity may offer more promise than breeding for immunity, and tests of hybrid progenies should be made with this in mind. A more

careful study of insect relationships seems desirable in breeding for resistance to insect-transmitted diseases.

Rankin (1927) was among the first to call attention to klendusity in relation to plant-disease control. He showed that certain varieties of red raspberry escaped infection under field conditions, although, when inoculated under controlled conditions, they were very susceptible. The rate of spread of mosaic in the field was also lower on the klendusic varieties than on the varieties preferred by the insect vectors.

This relationship has been investigated further by Schwartze and Huber (1937), who demonstrated a striking difference in susceptibility of red-raspberry varieties to *Amphorophora rubi* Kalt., the vector of raspberry mosaic (see Table III). These authors observed that the spread of mosaic appeared to be directly proportional to their relative aphid populations. The variety Lloyd George was shown to be so highly resistant that the aphids could not maintain themselves on it. In western Washington, this variety was never found infected with mosaic although graft inoculations demonstrated its susceptibility. In a later and more complete report, Huber and Schwartze

TABLE III.—FIELD COUNTS OF *Amphorophora rubi* KALT., MADE UPON TEN RED-RASPBERRY VARIETIES*

Variety	June 25		July 25	
	Range of aphid counts per cane	Av. no. aphids per cane	Range of aphid counts per cane	Av. no. aphids per cane
Antwerp.....	0-2	0.2	0-5	1.8
Chief.....	8-104	42.7	3-108	41.1
Cuthbert.....	0-16	5.0	0-30	10.8
Herbert.....	0-3	0.3	0-6	1.8
Latham.....	2-27	14.7	4-49	23.1
Lloyd George.....	0-0	0.0	0-0	0.0
Marlboro.....	1-9	4.6	4-42	16.8
Newburgh.....	0-6	2.0	0-9	4.6
Newman.....	4-17	10.1	1-26	12.5
Viking.....	0-39	16.0	3-83	32.8

* After Schwartze and Huber.

(1938) added the varieties Indian Summer, Pyne Imperial, and Pyne Royal to those on which *A. rubi* could not maintain its population. Reproduction was slow and the population remained small on Antwerp, Herbert, Marcy, and Newburgh.

The nature of the resistance to the aphids was not determined, although it was stated that "the behavior of *Amphorophora rubi* when confined to Lloyd George plants under cages indicates that resistance probably results from a lack of suitable food for the insect rather than the presence of an actively repellent substance." When the variety Lloyd George was crossed with susceptible varieties, the aphid-resistant character was inherited. The significance of a knowledge of these relationships in the breeding of improved varieties of raspberries is obvious. A true mosaic-resistant variety would be desirable; but if this is not easily obtained, a klendusic variety would be of great economic value. Even though only partial control could be obtained by the use of such varieties, the efficiency of control of mosaic through certification would be greatly increased by the slower rate of spread of the disease on klendusic varieties.

A similar relationship has been shown to exist among varieties of cranberry and the leaf hopper (*Euscelis striatulus*), vector of cranberry false blossom (Wilcox and Beckwith 1933). The degree of susceptibility of cranberry varieties under field conditions and the relative rate of spread of the disease were shown to be directly correlated with their attractiveness to the leaf hopper.

One of the best known examples of resistance to insect injury in plants is that of the native American grapes to the grape *Phylloxera* (*Phylloxera vastatrix* Planchon). This insect is a native of Eastern North America and commonly infests the various species of wild grape that, through natural selection, have acquired a high degree of tolerance to the insect. When the *Phylloxera* was introduced into France about 1860, the European species of grape proved to be so susceptible that within 25 years nearly one-third of the vineyards of France had been destroyed by it. The *Phylloxera* attacks both leaves and roots, but the greatest injury is caused by those which feed upon the roots. Numerous soft, watery galls are formed on the infested roots. These galls soon decay and greatly impair the root system, and the death of the entire plant is often the result.

Many control measures have been used in combating the *Phylloxera* on European grape varieties, but the most successful has been the use of certain species of American grape as resistant stocks on which to graft the desired European species. The mechanism of the resistance or tolerance of the American varieties is not known.

Searls (1935) has shown that varieties of peas with normal yellow foliage are more resistant to the pea aphid [*Illinois pisi* (Kalt.)] than those varieties with darker green foliage. It is not known whether the reaction is caused by the color alone or by some other factor associated with it. Because the aphids are the most common vectors of virus diseases, this discovery is obviously significant in the study of insects in relation to the transmission of virus diseases in general. It will be very interesting to know if other aphids react in a similar way to the color of the host plants.

An extensive study of the resistance of sorghums to the chinch bug has been reported by Snelling, Painter, Parker, and Osborn (1937). A wide variation in resistance was found, and the inheritance of the character was demonstrated. Resistance is due, not to a preference for the more susceptible varieties, but rather to the ability of the plant to grow or recover regardless of the continued feeding of the chinch bugs. The exact mechanism of resistance was not determined. These authors give a comprehensive review of the literature on insect resistance in plants.

The problem of inherent plant resistance to insects has been discussed at some length by Folsom and Wardle (1934), who cite numerous references bearing on the subject, and by Sweetman (1936). The latter, in considering the causes of resistance, groups them into three categories as follows:

1. Physical factors, such as thickness of epidermis and degree of pubescence.
2. Chemical factors; the presence in the plant of alkaloids, acids, tannins, or other chemicals.
3. Physiological factors, including vigor, quick recovery from injury, seasonal adaptation, early maturity, and copious sap flow.

Sweetman lists 66 examples of plant resistance to insects involving 47 different species or varieties of plant, and a some-

what larger number of insects. Of these, 17 were classed as caused by physiological factors, 10 by physical factors, 9 by chemical factors, and 8 by both physical and chemical factors, and 22 were classed as unknown.

The problem and its practical possibilities have been neglected somewhat by entomologists, as pointed out by Mumford (1931), who has discussed briefly some of the factors involved in insect resistance. He has proposed two new terms to express his concept of the nature of the resistance. Insect resistance caused by external protective agencies such as a thick epidermis or excessive pubescence is called *epiphyllaxis*. Resistance caused by internal factors such as the nature of the cell sap or protoplasm is called *endophyllaxis*. The latter category would include not only those characters that are unattractive or repellent but also the failure of the cell contents to meet the food requirements of the insect.

A very good discussion of the resistance of plants to insect injury was presented in 1924 by McCulloch, who stressed the need for a more fundamental study of the subject. Less extensive general discussions of the subject of plant resistance to insect attack have been published by Treherne (1917), Lees (1926), Parker and Painter (1932), McLeod (1933), and others. McCulloch recognized six kinds of specific cause of resistance: (1) habit of growth, (2) morphological characters of plants, (3) physiological characters of plants, (4) mechanical factors, (5) ability to recover from injury, and (6) external or environmental factors.

Insect resistance often escapes observation because the insect itself is not noticeably injurious, but it should be remembered that insect vectors of plant diseases may be very destructive indirectly through the diseases that they transmit. The inherent resistance of plants to insect vectors, therefore, should not be overlooked in the study of plant diseases transmitted by insects.

The demonstration by DeLong, Reid, and Darley (1930*a, b*) that bean plants sprayed with Bordeaux mixture may absorb enough copper to kill leaf hoppers feeding upon them calls attention to a new type of artificially induced insect resistance in plants. There has not been sufficient investigation of the possibility of this method of insect control to justify generalizations as to the extent of its application in the control of plant

diseases through control of insect vectors. It has possibilities enough to justify more extensive investigations.

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CHAPTER XIII

THE ANATOMY AND PHYSIOLOGY OF INSECTS IN RELATION TO THE TRANSMISSION OF PLANT DISEASES

The complexity of the relationship between insect vectors and the disease that they transmit varies widely. The association may be a simple, mechanical one or may involve a highly developed state of symbiosis. Even in the less complicated relationships, the association is often based on some particular adaptation in the morphology or physiology of the insect vector. It is evident that some knowledge of insect anatomy and physiology is essential for a satisfactory understanding of the nature of insect transmission of plant diseases. Certain aspects that are of particular significance in a study of insect transmission of plant diseases will be discussed briefly in this chapter. Emphasis will be placed on the anatomy and physiology of those orders of insect which include species of recognized importance as vectors. For a more complete treatment of insect anatomy and physiology, the reader is referred to the work of Snodgrass (1935), Comstock (1936), Imms (1934), and Weber (1930) and to the numerous other standard entomological texts.

THE EXOSKELETON

Insects are small boneless animals possessing a sclerotized exoskeleton instead of the bony endoskeleton of the higher animals. The exoskeleton protects the more delicate internal structures from desiccation and from mechanical injury, and it serves also as a very satisfactory framework for the attachment of muscles. The exoskeleton is made up of three essential layers: One layer of living cells, the *epidermis*, is covered externally with a nonliving and noncellular layer, the *cuticula*, and is bounded internally by a very thin layer of noncellular structure, known as the *basement membrane* (Fig. 202). The cuticula is a secretory product of the epidermis and consists chiefly of a nitrogenous polysaccharide known as *chitin*. It is soft at

first but becomes hard and tough by infiltration with a very resistant sclerotizing substance. The cuticula is composed of two layers, the outer, heavily sclerotized *exocuticula* and the inner, thicker, laminated *endocuticula*. A very thin surface layer known as the *epicuticula* is often present. The cuticula may be smooth and glistening or variously sculptured by striations, granulations, or other markings.

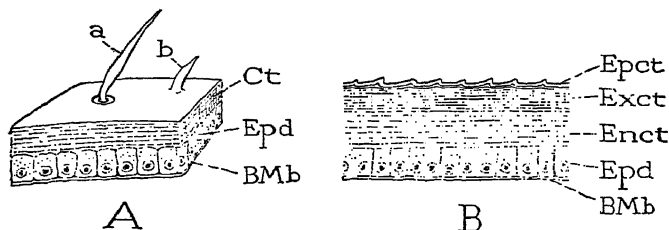


FIG. 202.—A diagrammatic representation of the structure of the body wall of an insect. A, a three-dimensional view of a section of the body wall bearing a seta (a) and a fixed hair (b); B, a vertical section; Ct, cuticula; Epd, epidermis; BMb, Basement membrane; Epct, epicuticula; Exct, exocuticula; Enct, endocuticula. (After Snodgrass.)

When the exoskeleton is concerned in the dissemination of plant pathogens, the process is generally of a mechanical nature. Dry spores, as well as wet sticky ones, adhere readily to the exoskeleton of insects, and they are likewise easily dislodged. Many kinds of fungus spore are disseminated mechanically in this way by insects that visit diseased and healthy plants. The effective surface for such mechanical dissemination of inoculum is greatly increased by the nature and abundance of cuticular processes. There are two general kinds of cuticular process: (1) The *setae*, or hollow pointed hairs, each produced by a modified epidermal cell, the *trichogen*. The setae usually have a membranous articulation at the base and a wide variety of shapes, structures, and functions. (2) The *microtrichia*, or fixed hairs, consisting of very small pointed extensions of the cuticula. These are fixed and solid and have no membranous articulation. They assume many shapes and sizes and are often very numerous. All these cuticular processes and the body appendages, such as legs, wings, and antennae, may serve as structures that catch and distribute spores and other kinds of inoculum (see Figs. 22 and 220). Inoculum that adheres to the

exoskeleton may be transported for long distances but usually, sooner or later, will be brushed off, either by accident or purposely by the insects. Many insects are clean in their habits and frequently rid their bodies of adhering particles by means of their legs.

THE MOUTH PARTS

Because feeding wounds made by insects are so important in disease transmission, the nature of the mouth parts assumes great significance in the study of insect structure from this point of view. Moreover, there is greater variation in the form of the mouth parts than in that of almost any other portion of the insect's anatomy. The variation in form is usually accompanied by a change in method of feeding. These differences in mouth parts and feeding methods have much to do with the relative effectiveness of different insects in transmitting plant diseases. Therefore, a knowledge of the different kinds of insect mouth parts is essential in a study of this kind.

Notwithstanding the great variation in structure, the mouth parts of most insects fall into one of two general types: (1) the mandibulate, or chewing mouth parts, and (2) the suctorial mouth parts adapted for piercing and sucking. There are, however, many modifications involving intergradations and overlapping of these two general types so that further division is desirable. We therefore recognize the following six types based on the methods of feeding: (1) chewing, (2) rasping-sucking, (3) piercing-sucking, (4) sponging, (5) siphoning, and (6) chewing-lapping.

The typical mouth parts of an insect consist of eight essential structures: the *labrum*, the *epipharynx*, a pair of *mandibles*, a pair of *maxillae*, the *hypopharynx*, and the *labium* (Fig. 203). These eight mouth parts vary so much in both form and function in different insects that they are often scarcely recognizable, but careful studies of their development and evolution have established their homologous nature.

1. Chewing Mouth Parts.—The chewing mouth parts are adapted for pinching off, chewing up, and swallowing bits of tissue. They are well illustrated by the mouth parts of a grasshopper (Fig. 203). The *labrum*, or the so-called "upper lip," fits over the mandibles in front and helps to pull the food into

the mouth. The *epipharynx* is a structure forming the ventral surface of the labrum and is continuous with the roof of the mouth and esophagus. Usually, it is equipped with organs of taste. The *mandibles*, or first pair of jaws, often called "teeth," are the structures that bite off and masticate the tissues. They

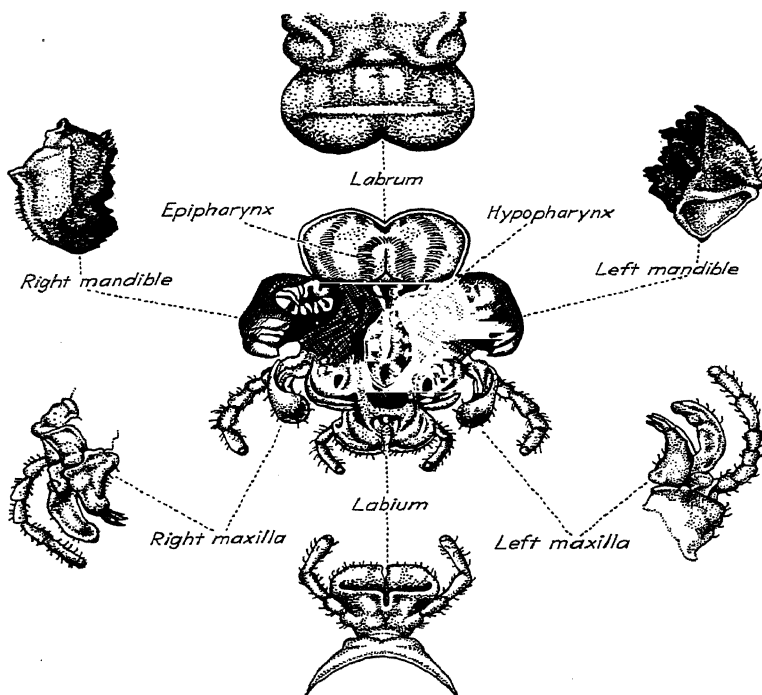


FIG. 203.—The mouth of a grasshopper, a typical chewing insect, showing the various parts. These fundamental structures are modified in the different species of insects to form mouth parts of widely different forms and functions. (Redrawn from Metcalf and Flint.)

are hard and usually equipped with sharp, pointed projections that make them effective grinders. Their chewing action is from side to side. The *maxillae*, or second pair of jaws, are more complicated in structure than the mandibles, each being composed of several articulated units (*cardo*, *stipes*, *palps*, *palpifer*, *galea*, and *lacinia*). Some of these work from side to side much

like the mandibles and aid in chewing. Others are antennaelike and are sense organs. The *hypopharynx* is a tongue-like prolongation of the floor of the mouth and is attached to the inside wall of the labium. The opening of the salivary duct is usually closely associated with the hypopharynx. The *labium*, or lower lip, is found opposite the labrum, or upper lip. It is composed of a third pair of jaws or appendages grown together along the mid-line and forming a lower lip. It has a pair of short antennaelike appendages, or labial palps, that also serve as sensory organs.

As representative of insects and chewing mouth parts may be mentioned grasshoppers, crickets, termites, and beetles. Many larvae, especially those of the Coleoptera and Lepidoptera, also have chewing mouth parts. These are the so-called grubs and caterpillars among which are found several important vectors of plant diseases.

2. Rasping-sucking Mouth Parts.—This type is intermediate between the chewing type and the piercing-sucking type and is represented by the mouth parts of thrips. Thrips feed upon growing plants and are known to be vectors of several destructive diseases. The mouth parts of various species of thrips have been studied by Borden (1915), Peterson (1915), Reyne (1927), and Wardle and Simpson (1927). The latter authors have described in considerable detail the mouth parts of *Thrips tabaci* Lind. and also its method of feeding on the tissues of the cotton plant.

The mouth parts of this insect are bent backward and underneath the head, forming a short cone-shaped proboscis (Fig. 204). The front part of the cone is made up of the *clypeus* and the *labrum*, separated slightly by a membrane. These structures are not symmetrical. The sides of the cone are formed by triangular structures probably derived from the *maxillae*, each bearing a three-jointed palp. The triangular *labium* composes the rear wall of the cone. It is longer than the labrum and maxillae and projects as a flexible flap. Both the labium and the maxillae are equipped with small hooks that probably serve to grip the leaf surface.

Four stylets enclosed within the cone are the actual piercing organs. The stylets are not symmetrical, there being two paired stylets, one on each side of a median stylet, and a single unpaired stylet to the left. The homologies of the stylets are

debatable; but, according to one interpretation, the paired stylets are probably of maxillary origin, the median stylet being derived from the *hypopharynx* and the left stylet being a *mandible*.

According to Wardle and Simpson (1927), the insect in feeding on a leaf surface, gashes an epidermal cell with a pickax-like movement of the single mandible. Further destruction of the

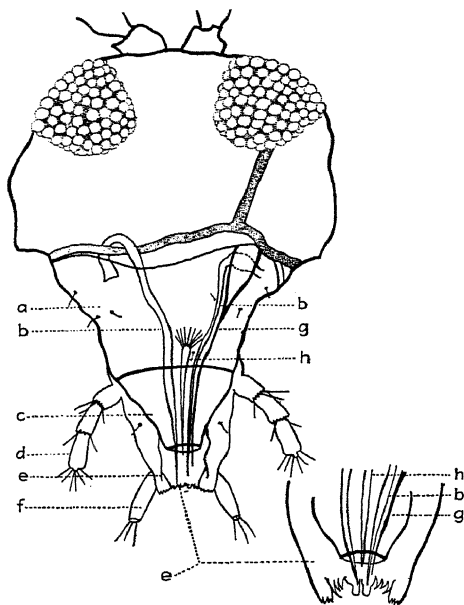


FIG. 204.—The assymetrical mouth parts of *Thrips tabaci*. *a*, clypeus; *b*, maxilla; *c*, labrum; *d*, maxillary palp; *e*, labium; *f*, labial palp; *g*, mandible; *h*, hypopharynx. (Redrawn after Wardle and Simpson.)

underlying tissue is accomplished by the more delicate, paired, maxillary stylets which can penetrate deeper into the tissues than the mandible can. The contents of the lacerated cells are sucked into the pharynx by applying the mouth of the cone to the leaf surface. The head and mouth parts of one of these insects in the act of feeding on the surface of a leaf is shown diagrammatically in Fig. 205.

Another type of the rasping-sucking mouth parts is found in the muscoid larvae (maggots) of the Diptera. The usual

structures are entirely suppressed in the maggots, being replaced by a pair of strong, sharp, chitinous "mouth hooks," movable as a unit in a vertical plane. These mouth hooks are often incorrectly called mandibles. They are solid cuticular structures that are shed with each molt but are in no way homologous with true mandibles. The entire facial region of the head is invaginated into the thorax, and a fold of the neck projects

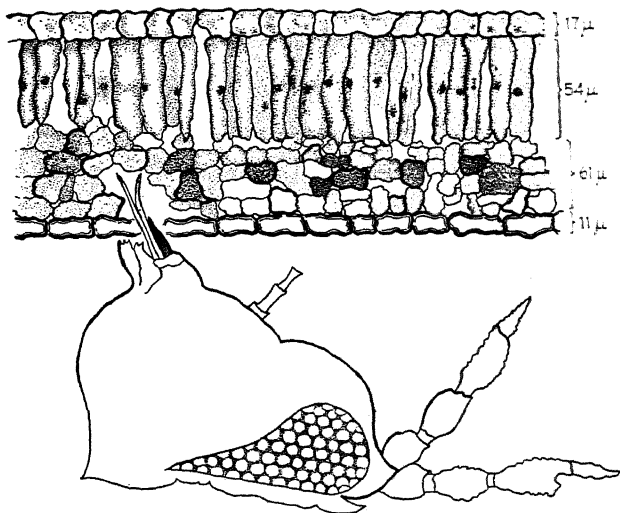


FIG. 205.—A diagram of the head of a feeding thrips indicating its size in relation to the leaf tissues and the depth of the feeding wounds. (After Wardle and Simpson.)

beyond the mouth to form a snoutlike structure that functions as the "head" of the maggot.

In feeding, the maggot lacerates the plant tissues or other substrate with the mouth hooks and, by means of a pump, sucks in the released liquid food. This more or less continuous rasping action of maggots that feed on plant tissues constitutes a very effective wounding agency from the standpoint of infection by plant pathogens. The symbiotic association of maggots with bacteria and other microorganisms that often are pathogenic to plants makes them assume a role of particular significance in this respect.

3. Piercing-sucking Mouth Parts.—The mouth parts of those insects which feed by piercing and sucking are highly specialized, and it is difficult to recognize the component parts as homologous with those of the chewing insects. There are many variations as represented by the aphids and plant bugs, the lice, the biting flies, and fleas. We are primarily interested in the Homoptera and Hemiptera as plant vectors, and our discussion of the piercing-sucking mouth parts will be limited to representatives of these groups.

The essential structures of the Hemipterous mouth parts are represented in Fig. 206. They consist of a slender beak of three or four segments surrounding and partly enclosing four extremely slender, sharp-pointed stylets so fitted together as to resemble a single bristle. These piercing stylets are often referred to as "mandibular" and "maxillary bristles" or merely as "setae." Since the latter term has been used extensively by those who have studied the mechanism of feeding of piercing-sucking insects on plant tissues, it is used in most of the following discussions.

The beak enclosing the setae is not a complete cylinder but has a groove along one side in which the four setae lie. This grooved structure is the *labium*. The four setae are *mandibles* and *maxillae*, the latter being the innermost two enclosed by the pair of mandibles. The inner surface of each of the maxillae is doubly grooved from end to end so that fitting together they form two closed tubes (Fig. 206A). The dorsal tube, usually the larger of the two, is the suction tube through which the plant juices are sucked. The smaller, ventral tube is the salivary tube through which saliva is ejected into the plant.

The two mandibles fit closely on opposite sides of the maxillae almost completely enclosing them but permitting a free sliding movement of the maxillae inside the enclosing mandibles. Near the base of the labium, on the open, or dorsal, side, the groove is covered by a short flap, the labrum. The hypopharynx lies between the bases of the maxillae. It is penetrated by salivary ducts that open into the salivary tube. It is heavily sclerotized but is small and inconspicuous.

The structure as described above may be considered as representative of the plant-sap-sucking insects of the orders Homoptera and Hemiptera. There is considerable variation in the details of structure among the different species of these orders,

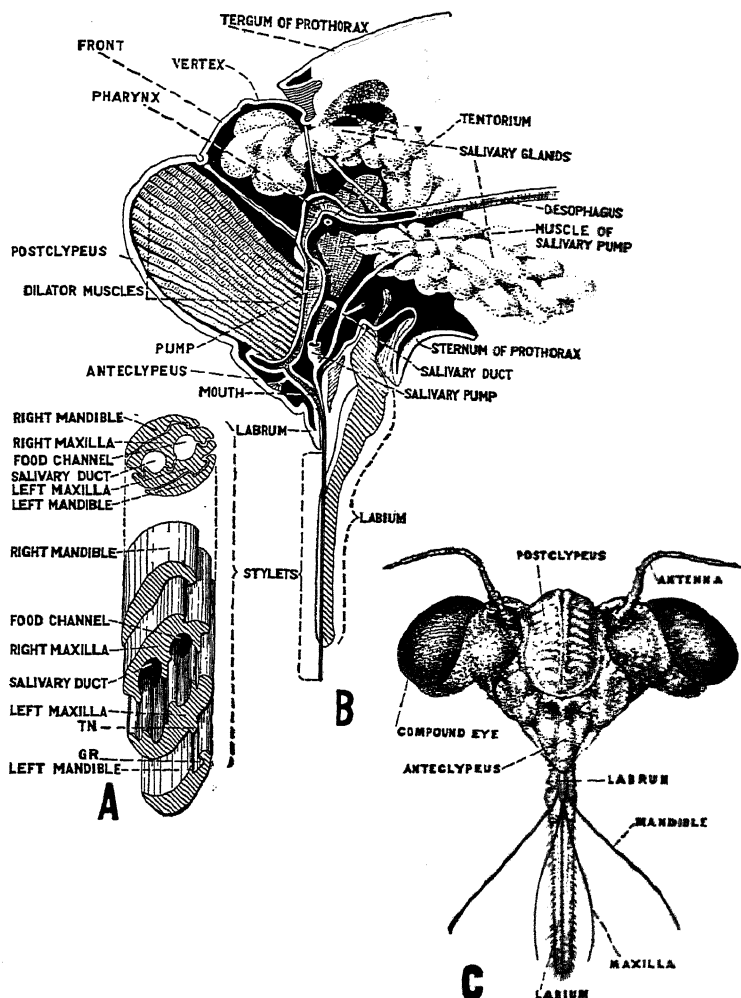


FIG. 206.—The piercing sucking mouth parts of the squash bug and cicada. *A*, cross section and isometric projection of mouth parts of the squash bug; *B*, a sagittal section of the head of the periodical cicada showing the mouth opening, the sucking pharynx, and the salivary glands; *C*, front or dorsal view of head and mouth parts of a dog-day cicada. (After Metcalf and Flint.)

but the fundamental arrangement of the mouth parts and their functions are essentially the same in all.

The detailed mechanics of feeding by the piercing-sucking insects has not been studied very extensively. The small size of the insects and the nature of their feeding have made the subject a difficult one. In relatively recent years, since the importance of sucking insects as vectors of plant viruses has been fully realized, the subject has received renewed attention. Earlier descriptions were brief and vague. It was stated simply that the slender stylets, using the grooved labium as a guide, were forced into the plant tissue by the protractor muscles at their base. However, when the feeding punctures (Figs. 223

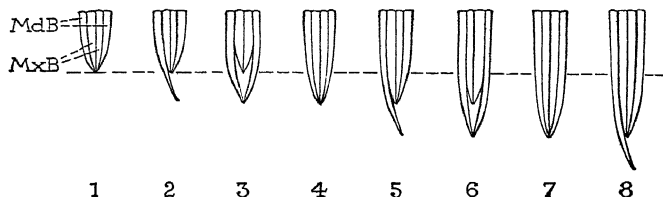


FIG. 207.—Successive stages in the insertion of the setae of Hemiptera according to Weber's interpretation. *MdB*, mandibular setae; *MxB*, maxillary setae. (After Snodgrass.)

and 224) are examined, it is evident that the range of action of the protractor muscles is not sufficient to force the setae to the depth that they reach in the plant tissues. Moreover, in many species (*Psyllidae*, *Coccidae*, etc.) the setae are much longer than the labium, and when the insect is not feeding the setae are retracted in a long flexible loop outside the labial groove. It would be physically impossible for the retractor muscles to force such long flexible setae into the plant tissues. The retraction of these long setae from the plant tissues would be difficult to explain; but Heriot (1934) has called attention to the fact, often overlooked, that the setae are cast off and renewed with each molt and that insects with very long setae, such as the scales and certain aphids, often never retract their setae after they once have been inserted deep into the tissues.

The mechanics of penetration by these insects has been investigated more extensively by Weber (1933), who has shown that the four setae are not moved simultaneously as a unit by the protractor muscles but are moved independently of each other.

According to this authority, the mandibular setae are the actual piercing organs and operate as follows: One mandibular bristle is forced out in advance of the remaining setae for a short distance by the protractor muscles, the sharp point of the single bristle making a small opening in the plant tissue. This operation is followed by a similar thrust by the opposite mandible after which the two enclosed maxillary bristles are lowered so that all four bristles assume the original position relative to one another but are slightly embedded in the surface of the plant tissue. By continued repetition of this procedure, which is

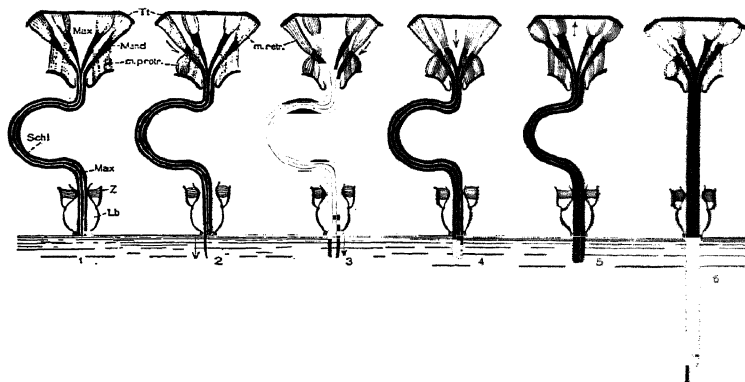


FIG. 208.—A diagrammatic drawing illustrating the mechanism of penetration of plant tissue by Hemipterous insects having long setae stored in a coil when retracted. 1, position of beak before penetration; 2 to 6, successive stages of insertion. (From Snodgrass after Weber.)

illustrated diagrammatically in Figs. 207 and 208, the setae are gradually driven deeper and deeper into the tissue until the required depth is reached. They are prevented from slipping back after each individual thrust, in some species by barbs on the mandibular setae and in others by the clasping action of the enclosing labium. According to Grove (1919), this clasping action may be due to increased pressure of body fluids; but Weber has shown that usually there are special labial clamps, equipped with appropriate muscles, for this purpose (Fig. 208).

There is considerable variation in the structure of the mouth parts of the different families of the Hemiptera, and further studies may reveal variations from the mechanisms described above; but, in any case, the piercing-sucking insects are capable

of penetrating plant tissues with a surprising degree of skill and are able to select the tissues most suited to their needs. For a further discussion of the feeding of the sucking insects from the standpoint of the plant tissues, see Chap. XV.

The sucking process begins as soon as penetration of the tissue has been accomplished. This involves a flow of saliva outward through the salivary canal into the tissues and a flow of the plant juices inward through the food canal. The saliva is forced through the salivary canal by the salivary pump, a muscular sac connecting with the salivary duct. The force required for the flow of the plant sap is provided, in part by capillarity and positive sap pressure in the plant, and in part by active suction produced by the dilation of the pharynx. Thus, a two-way flow through the beak is maintained during the feeding process: an outward flow of saliva and an inward flow of a mixture of plant sap and saliva.

The sucking insects are perhaps the most important group of insect vectors of plant diseases. The nature of their mouth parts and their mode of feeding make them effective agents of inoculation and ingression. They are especially effective in the transmission of virus diseases, and there appears to be a high degree of adaptation to virus transmission in many species of the Homoptera.

4. Sponging Mouth Parts.—This type is found in the common housefly and other nonbloodsucking Muscidae as well as in certain other families of the Diptera. In these, the *labium* is modified into a fleshy, elbowed, and retractile proboscis, the end of which consists of a pair of large spongelike organs, the *labella* (Fig. 209). These are traversed by a series of tubular channels, the *pseudotracheae*, opening by a narrow slit along the exposed side. The food channel is formed by the labrum, epipharynx, and hypopharynx. The mandibles are lacking, and the maxillae are modified into a pair of maxillary palps. Most insects with mouth parts of this type are unable to penetrate either plant or animal tissue; but there are some species in which the cleft between the labellar lobes is armed with several rows of small, sharp *prestomal teeth* with which tissue may be injured by a rasping action. The skins of animals are often lacerated by insects equipped with prestomal teeth, but the mechanism is not known to be used by any plant-feeding species.

When feeding on liquid matter, the pseudotracheae are brought into contact with the liquid and are filled by capillary action. The pseudotracheae all converge at a single point near the mouth of the food channel, and from this point the liquid is sucked into the esophagus. In feeding on solid material the labellum is placed on the food, and the liquid contents of the crop are regurgitated along with salivary secretions. This liquid dissolves the solid material and is again sucked in with the newly dissolved food.

This habit of regurgitation is of considerable significance in the dissemination of pathogenic microorganisms. It has been shown

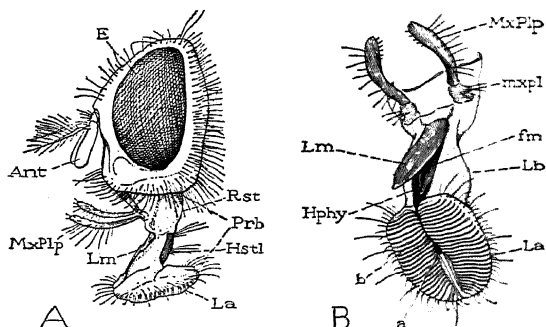


FIG. 209.—Head and mouth parts of the common housefly (*Musca domestica*). A, lateral view of head with proboscis extended; B, anterodistal view of proboscis showing the pseudotracheae (*b*) leading to the aperture (*a*) which leads to the food channel (*fm*) that lies between the labrum and the hypopharynx. (After Snodgrass.)

that the liquid contents of the crop of certain species of fly contain large quantities of viable bacteria among which pathogenic species are frequently found. It would be difficult to conceive of a more effective method of distribution of these microorganisms by insects than the one provided by this method of feeding, and there is considerable evidence that insects of this type are important vectors of several plant diseases.

5. Siphoning Mouth Parts.—The mouth parts of moths and butterflies are of the siphoning type. Only in rare cases are they able to penetrate plant or animal tissue. Such mouth parts are highly specialized for feeding on liquid food (Fig. 210). The labrum and the mandibles are usually absent or very greatly reduced, and the labium occurs only as labial palps. The

proboscis is formed by the two maxillae, the galeae of which are greatly elongated, each with a channel along its inner surface. The two grooved galeae are held together by hooks and interlocking spines, forming a long tubular proboscis through which

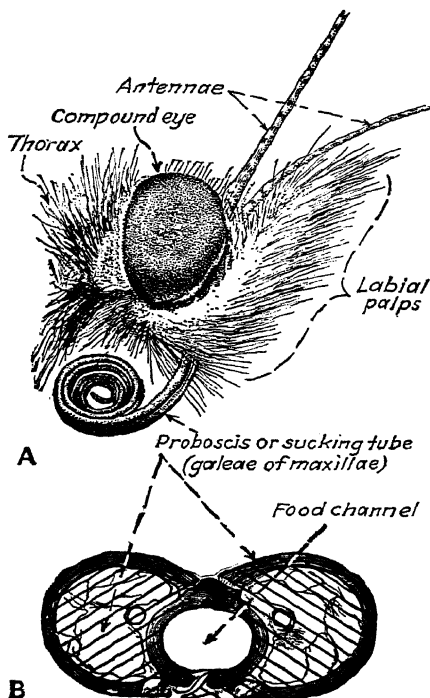


FIG. 210.—The siphoning mouth parts of a moth or butterfly. *A*, a side view of the head with proboscis partly coiled; *B*, a cross section of the proboscis showing the right and left galeae locked together to form a food channel. Insects with siphoning mouth parts do not make wounds in plant tissue but may act as vectors for plant pathogens that infect through the floral organs. (After Metcalf and Flint.)

liquid foods are imbibed. When not in use, the proboscis is coiled up like a watch spring. In feeding, it is uncoiled, and the tip is inserted into a liquid food such as the nectar of flowers. The siphoning mouth parts are characteristic of adult Lepidopterous insects (moths and butterflies). The larvae of these insects

(caterpillars), however, usually have mandibulate mouth parts adapted to chewing.

Insects with siphoning mouth parts act as vectors of plant diseases chiefly as a result of their regular visits to flowers in

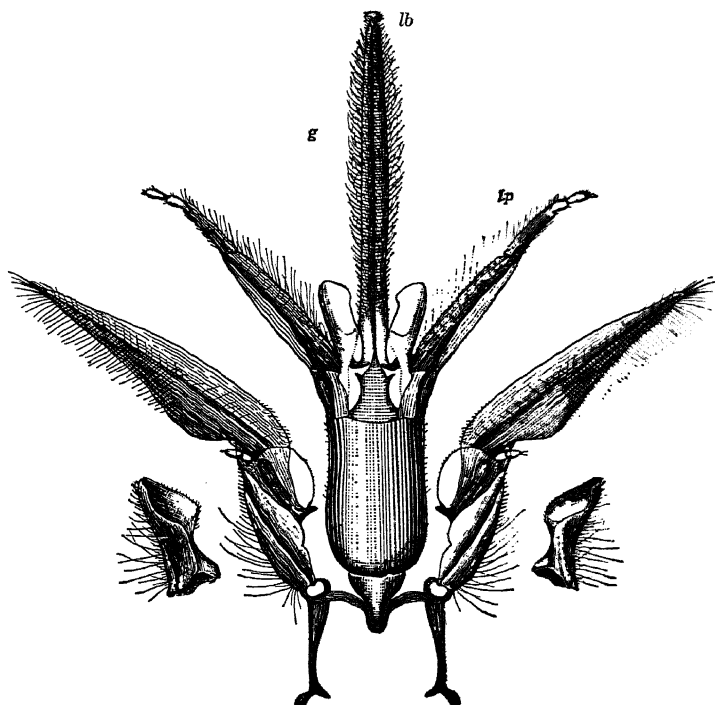


FIG. 211.—The mouth parts of the honeybee. Ventral aspect showing the grooved glossa (*g*) with the terminal spoonlike labellum (*lb*). In feeding position, the labial palps (*lp*) cover the grooved glossae, forming a suction tube through which the nectar passes. The labellum serves as a spoon to lap the nectar from relatively inaccessible places. (After Folsom and Wardle.)

search of nectar. The pathogens disseminated are those which find the flowers a favorable place for infection, and the inoculum is disseminated mechanically without wounding in all the known cases. The inoculum usually adheres to the setae or other bristlelike processes that serve also for the transfer of pollen grains.

6. Chewing-lapping Mouth Parts.—The honeybee and certain other species of Hymenopterous insects have complicated mouth parts representing a combination of the chewing type and a lapping tonguelike structure (Fig. 211). The labrum and mandibles are of the chewing type but are used more as tools in processing food than in feeding. The labium, however, has been modified to form a lapping tonguelike proboscis, the *glossa*. This structure is deeply grooved and densely covered with hairs except at its base, terminating in a small spoonlike lobe, the *labellum*. There seems to be a difference of opinion as to how these structures are used in feeding; probably, they are used in different ways. Easily accessible liquid foods are sucked into the mouth through a temporary food channel formed by the glossal tongue of the labium and the two labial palps. If the food is less accessible, suction is aided by a rapid lapping movement of the glossal tongue. In any case, the insects with mouth parts of the type represented by the bees, like those with siphoning mouth parts, are concerned chiefly with mechanical dissemination of inoculum that infects the floral organs. They make no wounds in the plants that are of importance in infection. Certain other Hymenopterous insects, such as the sawflies, have mandibles adapted for chewing and make wounds of various types on plant tissue. This is especially true of many of the Hymenopterous larvae.

THE SALIVARY GLANDS

Practically all insects are equipped with a pair of salivary glands generally located in the thorax, one on either side of the fore-intestine (Fig. 206*b*). The ducts from the two glands unite to form a common salivary duct with an opening between the labium and the base of the hypopharynx. There is often a saclike salivary reservoir opening off the salivary duct.

Relatively little is known about the exact functions of the salivary secretions, but they are known to vary considerably in the different kinds of insect. Their close association with the mouth parts obviously indicates that they are concerned in some way with the act of feeding. In the case of a number of plant-sap-sucking insects, it has been shown that the saliva possesses diastatic action, converting starch into soluble sugars. There is some evidence that the saliva has a solvent action, also, on

other cell constituents. Since the food canal is extremely small, often less than 1 or 2 microns in diameter, a low viscosity of the sap and the absence of occluding particles would be decidedly advantageous. In many sucking insects, a so-called "seral sheath" is formed around the embedded setae. This is thought by some to be largely of salivary origin and by its nature seals the opening, making it airtight, and increases the efficiency of the sucking operation.

The salivary glands appear to be in some way associated with the biological transmission of plant viruses by insects, but the exact nature of the association is not clearly understood. Inasmuch as the mouth parts of the piercing-sucking insects do not permit of the regurgitation of ingested plant sap, the virus particles ingested with the sap apparently find their way through the insect's body, accumulate in the salivary glands and are transmitted to healthy plants in the salivary secretions.

The salivary secretions of many insects are distinctly toxic to plant tissues, whereas those of other species apparently are harmless. The nature of the toxicity is not known, but it expresses itself in a variety of ways. A relatively large group of pathological effects, involving local injury or general physiological disturbances, is caused by insects that inject their salivary secretions into the plants. For a more complete discussion of this aspect of the problem, see Chap. V.

THE ALIMENTARY CANAL

The alimentary tract of the vector is involved to some extent in practically all cases of insect transmission of plant diseases, except in those consisting of simple mechanical distribution of inoculum. When transmission is biological, there is often a symbiotic relationship in which the alimentary canal and its appendages are structures of vital importance. Striking anatomical modifications of the digestive tract often occur as a result of symbiosis with microorganisms. A knowledge of the structure and function of this organ, therefore, would appear to be necessary for fundamental study of insect transmission of diseases. Insects of many different species regularly ingest quantities of inoculum of plant pathogens. Relatively little is known of the fate of the ingested inoculum and its significance in the spread of the disease concerned. In the following pages,

a brief description of the digestive system of insects will be given, with particular reference to those structures and functions which are potentially of significance in disease transmission.

The alimentary canal of insects is essentially a tube. It may be straight, or variously looped if its length exceeds that of the body. It is functionally a device for holding food in close contact with digestive and absorptive surfaces, having at one end a special organ of ingestion and at the other an organ of egestion. The digestive canal is composed of three primary divisions: (1) the *stomodaeum*, or fore-intestine; (2) the *mesenteron* (*ventriculus*), or mid-intestine; and (3) the *proctodaeum*, or hind-

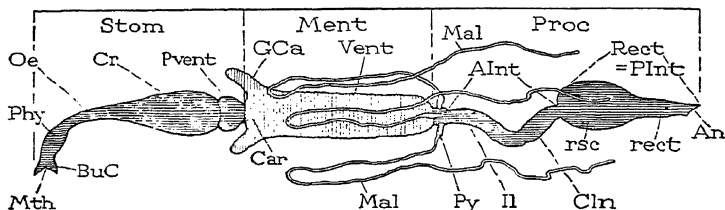


FIG. 212.—A diagrammatic and conventionalized alimentary canal of an insect showing the usual subdivisions and typical outgrowths. *Stom*, stomodaeum; *Mth*, mouth; *BuC*, buccal cavity; *Phy*, pharynx; *Cr*, crop; *Pvent*, proventriculus; *Ment*, mesenteron; *GCa*, gastric caecum; *Car*, cardiac valve; *Vent*, ventriculus; *Proc*, proctodaeum; *Mal*, Malpighian tubules; *Py*, pyloric valve; *AIInt*, anterior intestine; *Il*, ileum; *Cln*, colon; *Rect = PInt*, posterior intestine or rectum; *An*, anus. (After Snodgrass.)

intestine. These divisions are usually separated by valvelike structures; the one separating the stomodaeum from the mesenteron being known as the *stomodaeal*, or *cardiac valve*, and the one between the mesenteron and the proctodaeum, the *proctodaeal*, or *pyloric valve* (Fig. 212).

1. The Stomodaeum.—The stomodaeum, or fore-intestine, is of ectodermal origin and is formed in the embryo as an invagination from the outside. The stomodaeum, therefore, is lined with cuticula of the same origin as that of the exoskeleton. This cuticular lining is cast out and renewed each time the insect molts, a fact of considerable significance in a study of the possible survival of microorganisms in the insect body during metamorphosis.

In its simplest form, the stomodaeum is little more than a tube connecting the mouth with the mesenteron; but in most insects

it has become differentiated into three regions, the *pharynx*, the *crop*, and the *proventriculus*. In addition, an undifferentiated section between the pharynx and the crop may serve as an esophagus. In some insects, the section just within the mouth proper is distinguished from the mouth as the *buccal cavity*. The primary functions of the stomodaeum are largely mechanical, but some digestion often takes place in the crop where the food is subjected to the salivary enzymes as well as to gastric juices that flow forward from the mesenteron.

The *buccal cavity* is the oral part of the stomodaeum and usually is not differentiated sharply from the pharynx. In ants, there is a well differentiated *infrabuccal chamber* in the form of a spheroidal sac opening into the mouth cavity by a short constricted canal (Fig. 35). This chamber is used as a repository for any solid material ingested by the ant with its liquid food. The accumulated residue is thrown out as a compact pellet. It has been shown by Bailey (1920) and Leach and Dosdall (1938) that these infrabuccal pellets often contain viable fungus spores and that ants are by this means potential vectors of plant diseases. The infrabuccal chamber is used also by the fungus-cultivating ants for transporting cultures of the fungus when new colonies are established.

The *pharynx* lies just behind the buccal cavity, with strong dilator muscles attached to its walls. In insects with piercing-sucking mouth parts, the pharynx is a highly developed pump, or sucking device, by means of which the liquid food materials are ingested.

The *esophagus* is merely a narrow section of the stomodaeum between the pharynx and the crop. It has no function other than the conduction of food to the crop.

The *crop* may consist of a simple enlargement of the posterior region of the esophagus, but in some insects it occurs as a lateral diverticulum of the esophagus. The diverticulum usually forms a simple sac, but in the *Diptera* it takes the form of a long slender tube with a bladderlike enlargement at the end (Fig. 213). The primary function of the crop is that of food storage. Most insects feed excessively when food is available, storing the excess food in the crop to be passed later into the ventriculus for digestion. There is evidence, however, that some digestion also takes place in the crop through the agency of salivary enzymes and

digestive fluids that flow forward into the crop from the ventriculus. There is no conclusive evidence that enzymes are secreted by the walls of the stomodaeum or that an appreciable amount of absorption takes place through them.

The contents of the crop of the Diptera are usually liquid and contain large numbers of bacteria and other microorganisms that play a part in the digestion of the food. This is regurgitated along with salivary secretions when the insect is feeding on solid or semisolid material. The regurgitated liquids dissolve the

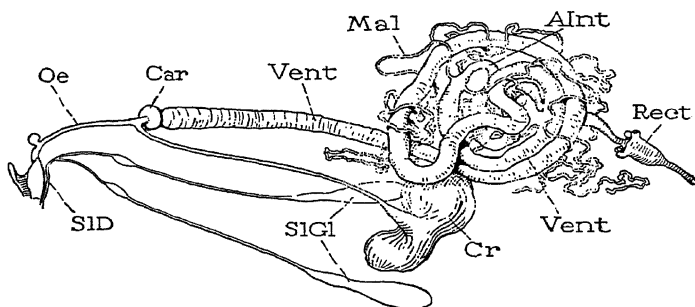


FIG. 213.—The alimentary canal and its appendages of the apple maggot fly (*Rhagoletis pomonella*) showing the diverticular crop characteristic of the Diptera. The liquid food in the crop usually contains quantities of living microorganisms. In feeding, the fly frequently regurgitates the contents of the crop, a habit that is conducive to the spread of pathogenic microorganisms. Oe, esophagus; Car, cardiac valve; SLD, salivary duct; SIGl, salivary glands; Cr, crop; Mal, Malpighian tubules; Vent, ventriculus; AInt, anterior intestine; Rect, rectum. (After Snodgrass.)

food materials and are then reingested. In this way, the surfaces fed upon are contaminated with whatever microorganisms are present in the crop. The soft-rot bacteria and the spores of the ergot fungi are disseminated in this way by several species of fly.

The *proventriculus* is the terminal region of the stomodaeum, and its structure and function vary in different insects. In its simplest form, it is a narrow tube more or less invaginated into the anterior segment of the mesenteron, forming the cardiac valve that serves to regulate the passage of food from the crop into the ventriculus. The structure is somewhat more complicated in chewing insects that feed on solid material. The walls are often heavily sclerotized and armed with toothlike spines

that serve to hold the food particles in the crop without completely closing the entrance into the ventriculus. In this way, the digestive fluids of the ventriculus may flow forward into the crop and partly digest the food before it enters the mesenteron. In some insects, there is evidence that the proventriculus also serves as an organ for further trituration of the food, a so-called "gizzard" or "chewing stomach."

2. The Mesenteron.—The mesenteron, or mid-intestine, is the middle segment of the alimentary canal and is the principal digestive organ of the insect. It is often called the *ventriculus*. It is generally in the form of a long tube of uniform diameter and is usually not differentiated into distinct regions. In some insects, as in the muscoid Diptera, the anterior portion is differentiated into a flat circular region containing the cardiac valve and separated from the rest of the mesenteron by a constriction.

The primary functions of the mesenteron are digestion and absorption. The epithelial layer of the walls of the mesenteron is much thicker than that of the stomodaeum. It is composed of large columnar cells of irregular length with ends projecting into the lumen. These are the digestive cells that secrete the digestive fluids and probably also absorb the digested foods. In some insects, there are two kinds of epithelial cell specialized for these two functions. The digestive cells are relatively unstable and are constantly disintegrating and being regenerated. The epithelium of the mesenteron is of endodermal origin and is not cast out when the insect molts.

The food contents of the mesenteron of many insects are enclosed and separated from the epithelium by a thin transparent membrane known as the *peritrophic membrane*, the function of which is somewhat obscure. It is usually interpreted as a chitinous intima of the mesenteron secreted by the epithelial cells. In the Diptera, it is the product of a ring of specialized cells encircling the cardiac valve. The membrane is permeable to the digestive fluids and to the digested food materials.

Blind pouches, or *caeca*, varying in number and shape are formed frequently as evaginations of the walls of the mesenteron. They most commonly originate at the anterior end near the cardiac valve but often arise from other parts, and in some species there may be several sets of caeca arising from different sections of the ventriculus (Fig. 214). The functions of these gastric

caeca differ in different insects. In certain insects, they are apparently secretory organs, but in others they act as receptacles for congenitally transmitted symbiotic microorganisms, Glasgow (1914), Buchner (1930), and others have studied the caeca and their enclosed symbiotic microorganisms. In the Heteroptera, the caeca with their symbiotic bacteria are especially common. These structures have assumed a position of importance in the study of insects in relation to plant diseases. Relatively little is known of the identity and function of the symbiotic microorganisms, but in several instances the caeca are known to harbor plant pathogenic species. The gastric caeca and their symbiotic

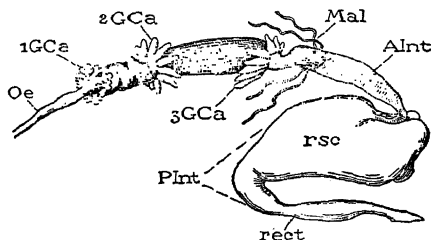


FIG. 214.—The alimentary canal of a scarabaeid larva (*Popilla japonica*) with three sets of gastric caeca (1GCa, 2GCa, and 3GCa). The gastric caeca usually serve as reservoirs of symbiotic microorganism which in some insects are plant pathogens. Oe, esophagus; Mal, Malpighian tubules; Ant, anterior intestine; PInt, posterior intestine; rsc, rectal sac; rect, rectum. (After Snodgrass.)

microorganisms should be studied more thoroughly from this viewpoint. For a more complete discussion of the question, see Chap. III.

The epithelial cells of the larval mesenteron are cast off and renewed by a layer of regenerative cells during metamorphosis. It is of interest to recall that, despite this renewal, bacteria may survive in the mesenteron of certain dipterous insects during pupation and ensure the contamination of the newly emerged imago (Leach 1933).

A striking modification of the alimentary canal occurs in the Homoptera, producing a structure known as the *filter chamber* (Fig. 215), formed by two distant parts of the digestive canal which are closely applied to each other and bound in place by a sheath of connective tissue. The parts bound together to form the filter chamber are usually the two ends of the mesenteron, often including also the anterior end of the proctodaeum. The

Homoptera are chiefly insects that feed on plant sap and, in order to get an adequate supply of the necessary nitrogenous food materials, usually ingest a great excess of water and soluble carbohydrates. The filter chamber is a device to permit the rapid elimination of the excess water and sugars which diffuse directly from the anterior region of the ventriculus into the proctodaeum, the nitrogenous and fatty foods being retained in the ventriculus for digestion and absorption. The sugar solutions eliminated in this way form the so-called "honeydew" commonly produced by many homopterous insects.

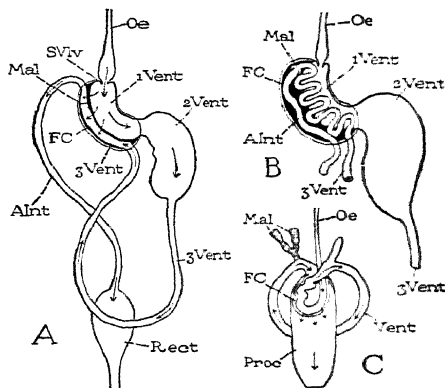


FIG. 215.—The filter chamber of Homoptera. A, B, and C, three types of differing degrees of complexity. (After Snodgrass.)

The honeydew, being rich in sugars, is a good medium for the growth of microorganisms and is concerned in the development of a number of injurious plant diseases. The so-called "sooty-mold" diseases are caused by fungi growing in honeydew that falls upon the leaves. The fungus growth is often so heavy that it interferes with the normal functions of the leaves, greatly injuring the plant. There is no evidence to show that the honeydew from viruliferous insects is of importance in the transfer of virus diseases. The virus particles apparently are unable to diffuse through the membrane of the filter.

3. The Proctodaeum.—The proctodaeum, or hind-intestine, like the stomodaeum, is of ectodermal origin, and its epithelium is cast out and renewed each time the insect molts. In its simplest form, the proctodaeum is a plain tube connecting the

mesenteron to the anus, but in most insects it is differentiated into several distinct regions. The point of union between the mesenteron and the proctodaeum is usually somewhat constricted, forming a *pyloric valve* that regulates the flow of materials from the mesenteron. The pyloric valve is generally of proctodaeal origin although, in some insects, it may be supplemented by a valve composed of mesenteric tissue, known as the *ventricular valve*.

Two main divisions of the proctodaeum usually are recognized: the *anterior intestine*; and the *posterior intestine*, or *rectum*. The former may be differentiated further into two regions on the basis of structure, these being separated by a constriction of the intestinal wall. The anterior segment is known as the *ileum* and the posterior as the *colon*. The primary function of the proctodaeum is to convey the residual products of digestion and the other waste products to the exterior; it is believed by some workers, however, that some absorption of foods takes place through the ileum. Absorption through certain modified regions of the proctodaeum of the termites appears to be the rule.

The *Malpighian tubules* empty into the anterior intestine near the pyloric valve. These are long, slender, blind tubes formed as diverticula of the proctodaeum and serve as excretory organs. They are immersed in the blood from which they remove the waste products of metabolism, especially urates and related compounds. In the larvae of some insects, the Malpighian tubules are modified to secrete a substance that is spun into silk threads with which the cocoon is woven. In other species, these organs function as caeca and harbor symbiotic microorganisms. The number of Malpighian tubules varies from 2 to more than 100 in different species of insects.

The *posterior intestine*, or *rectum*, is that portion of the intestinal canal immediately preceding the anus. It is composed of a dilated rectal sac and a narrow segment leading directly to the anus. The rectal sac is sometimes asymmetrically expanded into a *rectal caecum*. On the interior walls of the rectum are found six or sometimes three rectal pads, or "glands." They are outgrowths of the rectal epithelium composed of long columnar cells giving rise to protuberances into the lumen. The function of the rectal pads is not definitely known. Both secretory and absorptive functions have been attributed to them by different workers.

ORGANS OF REPRODUCTION

The reproductive organs of insects often are concerned directly in the transmission of plant diseases. They may be involved when the disease-producing agent is transmitted congenitally from one generation to the next in the vector or when the plant is inoculated by the vector in the act of oviposition. Insects, with very few exceptions, are bisexual, the male and female germ cells occurring in separate individuals; but, in certain species, males are rare or absent, and parthenogenesis is not uncommon. Since the male reproductive organs are rarely involved in the transmission of plant diseases, they are not included in the following discussion.

The majority of insect species are *oviparous*; i.e., the eggs hatch after oviposition. There are, however, many species in which the eggs hatch within the body of the female just before oviposition and the young are extruded as larvae or nymphs. This is called *ovoviviparous* reproduction. True *viviparous* reproduction occurs only in a few species, if at all. The method of embryonic development in the so-called viviparous reproduction of insects is fundamentally different from the viviparous reproduction of higher animals. Yet, in some species of insects, the young undergo extensive development within the body of the mother insect. For example, the larvae of the tsetse fly complete their development within the body of the female and pupate immediately after birth, being nourished before birth by the secretions of special glands.

Both oviparous and ovoviviparous reproduction occur in the Aphididae. In this family, ovoviviparous reproduction is usually parthenogenetic, the embryos developing in the egg tubes of the ovaries and being extruded as active nymphs. In oviparous reproduction, the spermatozoa are stored in a receptacle of the female (the *spermatheca*) at the time of copulation, and the eggs are fertilized individually as they are extruded from the oviduct. The embryo, therefore, develops outside the body of the female.

For convenience, the reproductive organs may be considered as composed of two parts, the *internal genitalia* and the *external genitalia*. The former harbor the germ cells, provide for their nutrition and development until they are ready for fertilization, and secrete the *chorion*, or shell. They also include devices for storing the sperm and ensuring fertilization as well as glands for

secreting a protective matrix that covers the eggs; and they discharge the eggs from the body at the proper time. The external genitalia provide means for the copulation of the sexes and include special structures that enable the female to deposit the eggs in a suitable place for their subsequent development and survival.

The *internal genitalia* of the female are made up of the following

essential parts: (1) a pair of *ovaries*; (2) two *lateral oviducts* arising from the ovaries and converging into (3) a *median oviduct*; (4) a *genital chamber*, or *vagina*; (5) *seminal receptacle*, or *spermatheca*, in which the sperms are stored; and (6) a pair of *accessory glands* (Fig. 216).

The *ovaries* usually consist of two groups of tapering tubes, the *ovarioles*, the number of which varies greatly in different species, four, six or eight in each ovary being the most common although they may be more numerous in certain groups. The germ cells are generated in the apex of the ovariole in the region known as the *germarium* situated just below the *terminal filament*. The germ cells pass into the larger part of the ovariole called the *vitellarium* where

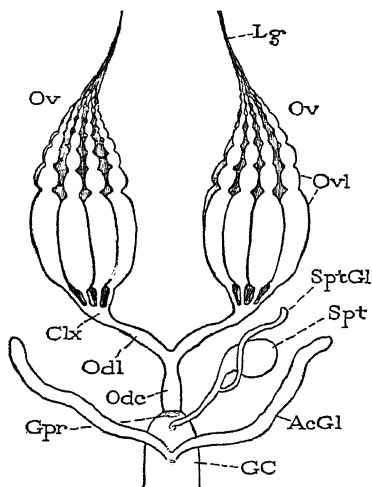


FIG. 216.—The structure of the female reproductive organs of an insect. *Lg*, ovarian ligament; *Ov*, ovary; *Ovl*, ovariole; *Clx*, calyx; *Odl*, lateral oviduct; *Odc*, median oviduct; *Gpr*, gonopore; *Spt*, spermatheca; *SptGl*, spermathecal gland; *AcGl*, accessory gland; *GC*, genital chamber (vagina). (After Snodgrass.)

they are nourished by specialized nutritive cells and are transformed successively into *oögonia* and *oöcytes* and into the fully developed but unfertilized egg. The epithelial layer of the wall of the vitellarium is composed of *follicle cells* that enclose each oöcyte in a definite sac, or *follicle*. These cells secrete the *chorion*, or egg shell; and when no nutritive cells are present, they also nourish the developing oöcyte.

Three general types of ovariole are recognized on the basis of the method of oöcyte nourishment. When there are no special

nutritive (nurse) cells, the ovariole is *panoistic*; when the nutritive cells are present, the ovariole is *meroistic*. In the panoistic type, the nourishment is elaborated by the follicle cells and is absorbed directly by the oöcyte. In the meroistic type, there are special nutritive cells that nourish the oöcytes by an active passage of the plasmic contents of the nurse cells into the cytoplasm of the oöcyte. In the meroistic type, the nutritive cells may be arranged alternately with the oöcytes (*polytrophic*) or they may remain in the upper part of the egg tube, retaining

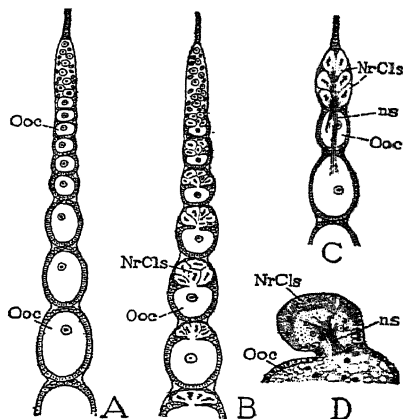


FIG. 217.—Three different types of ovariole based on the method of oöcyte nourishment. A, panoistic type; B, polytrophic type; C, acrotrophic type; D, upper end of acrotrophic ovariole of *Pseudococcus*. The method of egg formation and nourishment is of importance in the study of congenital transmission of plant pathogens by insect vectors. Ooc, oöcyte; NrCls, nurse cells; ns, plasmic strand. (After Shinji, from Snodgrass.)

connection with the oöcytes by long protoplasmic strands (*acrotrophic*) (Fig. 217).

The nature of the nourishment of the oöcytes is of particular interest because in cases of congenital transmission of symbiotic microorganisms the actual transmission occurs at this stage. "Infection" or contamination of the developing egg often takes place through the nurse cells or by direct invasion of the oöcyte. The process has been described by Brain (1923), Granovsky (1929), and others. The problem of congenital transmission of viruses by the insect vector has assumed renewed importance by the proof of such transmission of the virus of the dwarf

disease of rice in the insect vector *Nephotettix apicalis*. Since some viruses are transmitted through the egg and others are not, this stage of insect reproduction will be of critical significance in a study of insect transmission of viruses.

The two *lateral oviducts* are ducts into which all the ovarioles converge and into which they empty their eggs. In some species, the lateral oviducts are distended into large pouches (egg calyx) for temporary storage of eggs.

The two lateral oviducts unite to form the *median oviduct* which opens into the vagina through a constricted opening, the *gonopore*. The gonopore regulates the discharge of the eggs from the oviduct into the vagina.

The *vagina* is a cavity formed by an invagination of the body wall and has a chitinous lining. It functions as a copulatory pouch during mating and is sometimes known as the *bursa copulatrix*. It is continuous with the median oviduct, and the spermatheca opens into it dorsally near the anterior end. The external opening of the vagina is the *vulva* and serves both for copulation and for the discharge of the eggs.

In some insects, the *bursa copulatrix* provides a place for the development of symbiotic microorganisms that are congenitally transmitted by infecting the eggs as they pass out in oviposition (Buchner, 1930). In other species, the vagina and the rectum converge so that the posterior regions of the vulva and the anus are essentially identical. This modification has been shown, by Buchner (1930) and others, to be an adaptation to ensure the contamination of the eggs with symbiotic microorganisms from the intestinal tract. In insects with this modification, the microorganisms usually are harbored in a number of irregular pouches in the dorsal wall of the rectum. The egg in passing through the oviduct presses against the pouches and forces the microorganisms out onto the surface of the egg. This condition is well illustrated by *Dacus oleae* and its symbiotic bacteria described in Chap. VI (Figs. 88, 89, and 90).

The *spermatheca* is a receptacle for storage of the sperms received during copulation. The sperms are ejected from the spermatheca upon the eggs as they pass from the oviduct. It is primarily an invagination of the ectodermis and is lined with chitin. It is essentially a single organ but may be variously branched. It is usually saclike and is connected with the vagina by a slender

tube. A *spermathecal gland* is usually associated with the spermatheca. It is formed as a diverticulum of the spermathecal duct and secretes a fluid in which the sperms are discharged. In some species of insect, symbiotic microorganisms are found in the spermatheca along with the sperms, and congenital transmission is affected by contamination of the egg at the time of fertilization (Buchner 1930, Mansour 1934a).

The *accessory glands* are paired structures opening into the vaginal chamber through its dorsal wall. The glands usually

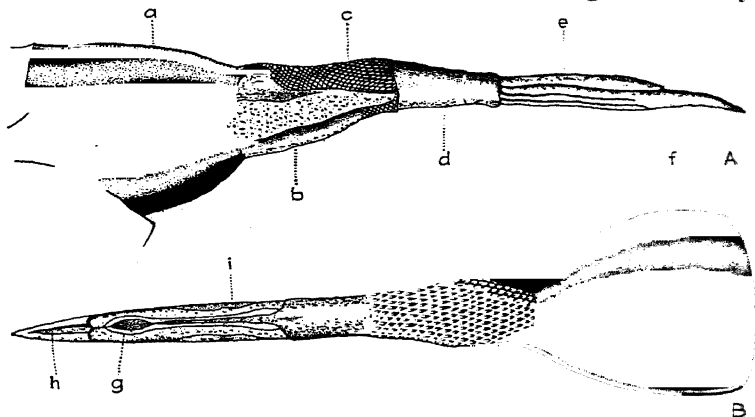


FIG. 218.—The ovipositor of *Rhagoletis pomonella*, the apple maggot fly. The sharp point of the ovipositor makes a puncture through the skin of the apple through which the eggs are inserted. Along with the eggs are inserted pathogenic bacteria that cause a decay of the ripe fruit and that also aid in the nourishment of the larvae. See Figs. 92 and 93. A, lateral view of extended ovipositor; B, ventral view; a, seventh abdominal segment; b, pleural membrane; c, basal sheath; d, distal sheath; e, ventral process; f, dorsal process; g, opening of cloaca; h, ventral groove; i, lateral groove. (After Dean.)

secrete an adhesive substance for attaching the eggs to the substrate or for gluing several eggs together in a single mass. They are often called *colleterial glands*. In the stinging Hymenoptera, one of the accessory glands secretes the toxic substance associated with the stinging apparatus.

The *external genitalia* of the female insect consist of structural adaptations for the deposition of eggs. Collectively, they constitute the *ovipositor* and are derived from appendages of the eighth and ninth abdominal segments (Fig. 218). The ovipositor enables the insect to deposit her eggs in the most suitable place

for hatching and for the subsequent development of the larvae. This organ is of particular interest to the student of insect transmission because it is used by many insects for inserting eggs into the tissues of plants (Fig. 90). The wounds made in oviposition are frequently the avenues of infection of plant pathogens. The oviposition wounds are especially important when the insect is also an agent of dissemination of the inoculum. The oviposition wounds made by the snowy tree cricket and by the apple-maggot fly function in this way (see Chaps. VI and VII). In many insects, the ovipositor is absent or greatly reduced, and the eggs are extruded and attached to smooth surfaces or introduced into naturally occurring cracks or crevices.

The size and shape of individual parts of the ovipositor vary greatly with different species according to the place of oviposition and the work to be done by the ovipositor. In most of the species that insert their eggs in the tissues of plants, the parts are provided with sharp sclerotized points that are well adapted for making the necessary punctures. There are some insects, however (as the Cerambycidae), which make the necessary puncture with their mouth parts after which the eggs are inserted with the ovipositor.

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CHAPTER XIV

THE INOCULA OF PLANT PATHOGENS IN RELATION TO INSECT DISSEMINATION

Plant pathogens produce many kinds of inoculum which are disseminated in many different ways. A consideration of the various kinds of inoculum and their adaptability to insect dissemination is helpful in a study of the relation of insects to plant diseases. Some kinds of inoculum are well adapted to wind dissemination, and others seem to be poorly suited for dispersal by wind but especially adapted to insect dissemination; still others may be adapted to dissemination by water or other agencies. With increasing knowledge of the methods of spore dispersal in fungi, it is even more evident that the various specialized methods of spore formation and liberation are very closely correlated with some equally effective means of dissemination. The nature and method of production of inoculum by a pathogen usually indicates clearly its most frequent method of dissemination and often leads to a better understanding of the disease.

Inoculum may be defined as that part, or developmental stage, of a pathogen capable of initiating a disease when placed upon the infection court under the proper environmental conditions. Spores of fungi and vegetative cells of bacteria are the most frequently observed units of inoculum although other structures may sometimes function. Inoculum may have important functions other than the initiation of a disease. Dissemination of the pathogen from place to place is one function, and consequently inoculum is usually composed of very small units that are easily disseminated by various agencies. Survival during periods of unfavorable environment and subsequent reproduction is another important function of inoculum. Therefore, much inoculum consists of units that are resistant to various unfavorable conditions such as desiccation, bright sunlight, and high or low temperatures. A third important function is that of multi-

plication. Very rapid multiplication is accomplished by the production of extremely large quantities of inoculum by a single individual.

Plant pathogenic fungi may reproduce either sexually or asexually. Some of those which reproduce sexually are homothallic, both male and female gametes being produced on the same mycelium. Others are heterothallic, each individual forming only one kind of gamete. Heterothallic plant-pathogenic fungi usually produce spores which on germination give rise to haploid mycelium that must fuse with another mycelium of

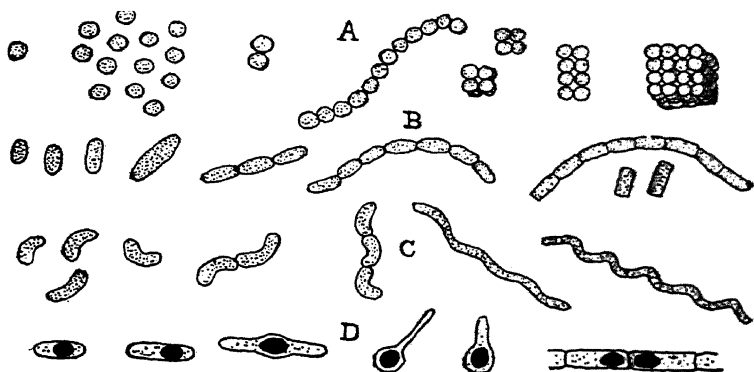


FIG. 219.—A diagram of the three morphological groups of the true bacteria. A, Coccus type; B, Bacillus type; C, Spirillum type; D, bacterial spores. Practically all the plant pathogenic bacteria belong to the Bacillus type. Spores are of very little significance as inoculum in bacterial diseases of plants. The vegetative cells constitute the principal inoculum. (After Heald.)

complementary sex before completion of the entire life cycle. Thus spores may, in some cases, serve as gametes as well as inoculum.

The most important plant pathogens fall within the following four groups: (1) bacteria, (2) fungi, (3) protozoa, and (4) filterable viruses. The inocula of the different groups will be discussed separately.

Bacteria.—The morphology of the plant pathogenic bacteria is relatively simple, and only a few different kinds of inoculum are produced (Fig. 219). The vegetative cells of bacteria constitute the most common inoculum; only a few less important bacterial plant pathogens produce spores although many species

of bacteria that are not pathogenic to plants produce them abundantly. Some of the plant pathogenic bacteria are said to have a filterable stage in which the organisms survive in units of submicroscopic size, but the significance of this stage of the bacteria, as inoculum, is not well known.

From the standpoint of size, the bacterial cell is well adapted to insect dissemination. An average cell of the bacterial plant pathogens would measure less than 1 micron in width and not more than 2 microns in length, being small enough to pass readily through the mouth parts of practically all plant feeding insects, even those with special structures for screening out solid materials. The size of the food channel of the small sap-sucking Homopterous insects is not a limiting factor; for it is rarely, if ever, less than 1 micron in diameter and is usually much larger.

The vegetative cells of plant pathogenic bacteria are variable in their resistance to unfavorable environmental factors, but they are more hardy than their simple structure and the lack of resistant spores would lead one to expect. Almost all plant pathogenic bacteria develop in an exudate or ooze composed partly of the plant sap and partly of products of bacterial metabolism (Fig. 67). It has been shown in numerous cases that the exudate has a decided protective effect on the bacterial cells. This is especially true in respect to desiccation. When they are subjected to dry conditions, the exudate surrounding the individual cells acts as a hydrophilic colloid having a high water-holding capacity that prevents injury by excessive desiccation.

Bacterial exudate is usually of a sticky consistency so that it adheres readily to the body of insects coming in contact with it. In this way, bacteria are disseminated on the exoskeleton of the insects and are not in danger of being destroyed by desiccation. Moreover, bacterial exudates often give off odors attractive to insects, and many exudates have much food value and are eagerly ingested by insects. These properties are all conducive to the frequent dissemination of bacterial plant pathogens by insects.

The fate of bacteria ingested by insects has not been extensively studied. However, the available evidence shows clearly that it is not always the same for all. Some bacteria are killed and digested in the alimentary canal of insects; others are more

resistant and survive, to be passed out in a viable condition in the excreta; still others are adapted to life in the insect body where they grow and multiply. It has been demonstrated that several species of bacteria that are pathogenic to plants are able to survive passage through the intestinal tract of their insect vectors. *Erwinia carotovora* and the seed-corn maggot, *E. amylovora* and fruit flies, *E. tracheiphila* and the cucumber beetle, and *Phytophthora savastanoi* and the olive fly are a few of the better known examples of bacterial plant pathogens that survive passage through the alimentary canals of their respective vectors. Although it is known that some bacteria thrive in the intestinal tracts of insects and others do not survive, very little is known about the factors that influence survival. This should be a promising field of investigation. The ability to survive and grow in the body of insects obviously is an adaptation favorable to insect dissemination and is a point that should not be overlooked in the study of vectors of bacterial diseases.

Insects, in addition to disseminating the bacteria, may provide them with a considerable degree of protection against environmental conditions. As pointed out in Chap. III, many bacteria, including some plant pathogens, live in a state of mutualistic symbiosis with insects, a condition that is especially conducive to insect dissemination. Bacteria that are adapted to survival in the insect's body during metamorphosis or hibernation have a distinct advantage over similar forms that are subjected to the more variable and hazardous environment elsewhere.

Spores are so rarely formed by plant pathogenic bacteria that it is scarcely necessary to consider them here. Their small size and extreme resistance to the environment would make them well suited to either wind or insect dissemination.

Pleomorphism is now recognized as a normal character of many bacteria. Filterable stages in which the bacteria exist in extremely small ultramicroscopic particles are not uncommon among plant-pathogenic species. It is not known that these filterable forms have any direct bearing on the problem of insect transmission; but, in examining insects for the presence of plant-pathogenic bacteria, failure to find visual evidence of the usual rod forms should not be considered as final proof of the absence of bacteria. Tests should be made for the presence of the pathogen in filterable form.

When all these factors are considered, it is evident that bacterial plant pathogens have many attributes that favor dissemination by insects. Several of the most destructive bacterial plant diseases are dependent almost entirely upon insects for transmission, and insects are known to spread others to a lesser extent. The methods of dissemination are still obscure for many others. Any insect that is a fairly constant visitor to plants affected with bacterial diseases should be looked upon with suspicion.

Fungi.—Three general types of inoculum are formed by fungi: vegetative mycelium, sclerotia, and spores. Vegetative mycelium as inoculum is not particularly well adapted to insect dissemination. It may serve as inoculum when it survives in seeds or plant parts used in vegetative propagation. The mycelium in plant parts that are eaten by insects is potential inoculum. Taubenhaus and Christensen (1936) have shown that *Fusarium vasinfectum*, the cotton-wilt pathogen, may be disseminated in the fecal pellets of grasshoppers and other insects that ingest infected tissues. The same authors (1936), on the other hand, have shown that neither the spores nor the mycelia of *Phymatotrichum omnivorum* survive passage through the intestinal tract of three species of soil-inhabiting insects. Larvae of *Phyllophaga* sp. and adults of *Blaptinus fuscus* and *Harpalus* sp. were fed on vegetative mycelium and on spores of the fungus, but viable cultures could not be recovered from the intestinal contents or from the fecal pellets. Insects that feed in decaying wood are potential vectors of vegetative mycelium that may serve as inoculum although there has been very little study of the possibility.

Many fungi form dense compact aggregates of hyphae filled with oil globules and other reserve foods. These structures are called "sclerotia," and they function chiefly as storage organs to aid in surviving unfavorable periods but also serve as inoculum, frequently being disseminated with seed or other plant parts. Because of the large size of most sclerotia, they are poorly adapted to insect dissemination and must depend on other agencies.

Spores are the most common type of fungus inoculum. There is a marked diversity in the kinds of spore formed, and many kinds are extremely well adapted to insect dissemination. A

fungus spore consists of one to several specialized cells and serves as a unit of reproduction and dissemination. Spores are usually resistant to the environment and aid in the survival of unfavorable conditions. They vary in size from approximately 1 to 200 or 300 microns in diameter, although the majority would probably measure less than 25. In size, as well as in many other respects, fungus spores are similar to pollen grains and are equally well adapted to insect dissemination.



FIG. 220.—The adherence of dry spores to the body of an insect. A, a leg of a fly that was brought in contact with a quantity of dry fungus spores on a glass slide, showing the spores adhering to the bristles; B, a single bristle more highly magnified, showing the loose attachment of individual spores; C, a leg from the same fly before contact with the spores.

Spores adhere readily to the legs, wings, bristles, and other parts of the insect body. Some spores are borne in a sticky liquid matrix that may facilitate adherence to the bodies of insects (Figs. 43, 99, and 221), but dry spores will adhere almost as well. This fact may be readily demonstrated by bringing the leg or wing of an insect in contact with dry spores of any common fungus and observing it under the microscope (Fig. 220). Not only do the spores adhere readily to the insect organ, but also they may be brushed off easily. The force responsible for the adherence of spores in this way is not clear although it may be in part the attraction of the thin films of water that are

known to exist on the surface of apparently dry bodies. It has been shown that it is not entirely a matter of electric charges. This phenomenon of adherence of dry spores to dry surfaces is discussed at some length by Hanna (1924) in connection with the dry-needle technique of picking up single spores. Hanna demonstrated that dry spores would adhere readily to dry needles regardless of the electrical charge of the needle. The force of adherence is not great, and the spores are easily dislodged. Whatever the explanation may be, the ready adherence of dry spores to dry surfaces adds to the ease with which they are disseminated by insects.

Fungus spores are notably resistant to environmental conditions. The resistance may be caused by the nature of the protoplasm, by a thick wall, by a hydrophilic sheath, or by a hydrophilic matrix in which they are borne. Many fungus spores are resistant also to the environment found in the intestinal canal of insects. In the majority of cases where the question has been investigated, spores have been found to pass through the intestinal tract of insects uninjured. One significant exception was pointed out by Taubenhaus and Christensen (1936) who fed *Fusarium vasinfectum* to a large number of insects. The fungus passed uninjured through all the phytophagous insects but did not survive passage through any of several mycophagous insects. This would suggest that the digestive juices of insects which normally feed on the tissues of higher plants are less injurious to fungus spores than those of insects which commonly feed on fungi.

A significant relationship between insects and fungus spores was reported by Schneider-Orelli (1911), who found that spores of the ambrosia fungus associated with *Xyleborus dispar* would not germinate when taken directly from the tunnels in the wood but germinate readily when recovered from the crop of the beetle. It was suggested that something in the intestinal contents of the beetle served as a stimulus to germination. A period of dormancy during which spores will not germinate is common for many fungi. The factors responsible for breaking the dormancy are obscure. Should the fungus be closely associated with an insect, it would not be unreasonable to look for the stimulus in the insect's body. Similar relationships between fungi and higher animals have been described by Massee and

Salmon (1902) and Buller (1934) in their studies of the coprophilous fungi. Many of these fungi which are normally found only on dung produce spores that do not germinate until they are eaten by animals and are passed out in the dung where they germinate readily and abundantly.



FIG. 221.—Stinkhorn fungi. A, a typical sporophore of *Dictyophora duplicata* showing the shining black sticky mass of spores and the lacelike veil; B, a fly feeding on the sticky mass of spores of *Mutinus curtisii*. The stalk of this fungus is bright rosy red, the spore mass is brown.

It is not necessary here to describe the many different kinds of spore formed by fungi as this information can be obtained from any standard mycological text. It will be sufficient to consider a few examples in which the methods of spore production and liberation show some special adaptation to insect dissemination.

A number of pathogenic fungi form their spores in a matrix that is attractive to insects. *Claviceps purpurea*, the ergot fungus, produces its conidia in a drop of sugary solution that is

eaten greedily by many insects, especially flies which also visit healthy flowers in search of pollen, thus serving as effective vectors of the fungus. The sugary solution has a characteristic odor that seems to attract the insects. These characters must be considered as adaptations on the part of the fungus that ensure insect dissemination of its spores.

A similar adaptation is shown by the stinkhorn fungi, which produce their spores in a sticky matrix with a foul, carrionlike odor (Fig. 221). Flies are attracted to the matrix, feed upon it, and effectively disseminate the spores that adhere to their bodies. The spores may pass uninjured through the digestive tracts of flies, and they are probably disseminated also in this way. This adaptive relationship has been studied and discussed at considerable length by Fulton (1889) and Cobb (1906, 1909). Fulton described the morphology and growth habits of a large number of these fungi and made extensive observations and experiments dealing with spore dissemination by insects. In concluding his discussion he states:

In the Phalloidei then it can scarcely be doubted that we have a group of fungi which have undergone great modification so as to become adapted for the dispersion of their spores by the agency of insects and especially by those which habitually affect putrid substances, and would therefore deposit the spores on the most suitable nidus for their growth. It is an example of the substitution of the more certain action of insects for the uncertain action of the wind, just as in the case of the cross-fertilization of flowers, where the former agency has so largely replaced the latter.

The chief adaptations shown by the Phalloidei in this respect were summarized as follows:

- (1) The formation and maturation of the spores within a closed volva, usually subterranean and protected largely from injury.

- (2) The sudden emergence and expansion of the receptacle, not by the comparatively slow process of cellular growth, but by a mechanical springlike apparatus acting quickly.

- (3) The freely exposed hymenium undergoing rapid mimetic changes, whereby putrid matters are simulated in color, consistency, and usually odor.

- (4) The development of a striking form, and of such colors as will make the receptacle most conspicuous, and the production of accessory parts for the same purpose.

(5) The rapidity of the whole cycle of phenomena by which the risk of accidental destruction, by animals, etc., is lessened, and the effective dispersion of the spores secured.

Fulton concluded his discussion with the following significant remarks concerning the teleology of putrefactive odors in general:

I may finally add a word as to the importance of these observations that flies and other insects transport living spores of fungi in relation to the transport of bacteria. The teleology of the bad odors of putrefaction has, so far as I am aware, never been explained; but since these odors depend upon the presence of organisms, and serve to attract multitudes of flies, and since it is a rule in Nature that no organism aids another except, so to speak, with the object of ultimately benefiting itself, it seems to me highly probable that the odor of putrescent matter has been developed in relation to the visits of flies. The advantage to the bacteria is obvious, for by the odor produced, the irregular fortuitous action of the wind is largely replaced by the direct purposive action of insects; and hence, once the odor is developed, flies will form the chief means of dispersal. It has been shown above that spores may retain their vitality during their passage through the digestive canal of flies, and there can be little doubt the same applied to bacteria. Flies may thus serve to disperse not only the microorganisms associated with putrefaction, but also those which are the cause of certain diseases, such as anthrax, etc., in which the products are attractive to them; and since flies, apart from their own powers of locomotion, are known to be carried long journeys in railway trains and steamers, they may serve to carry infection and spread diseases to distant places.

Cobb (1906, 1909) has elaborated on this phenomenon of spore dispersal by flies and has supplied additional evidence based on observations and experiments with *Ithyphallus coralloides*, a stinkhorn fungus that he erroneously thought to be the cause of a root rot of sugar cane in Hawaii. Cobb showed that the spores of this fungus were not injured by passage through the digestive tract of flies. He showed by actual spore counts that a single "flyspeck" often contained as many as 22,400,000 spores of the fungus.

The blue-stain fungi (*Ceratostomella* spp.) appear to form their spores in a manner that makes them better adapted to dissemination by the bark beetles than by any other means. Both the conidia (*Graphium*) and the ascospores are produced under the bark in sticky solutions that make them unsuited to wind dissem-

ination (Figs. 102, 103, and 104). The most abundant sporulation occurs in the tunnels and pupal chambers of the bark beetles, the sporophores and perithecia pointing toward the center of the tunnels in such a way that the spore masses are in the most advantageous position for coming in contact with the insects (Leach, Orr, and Christensen, 1934).

Many Ascomycetes liberate their ascospores by forcibly ejecting them from the asci. This process has been described fully by Buller (1909, 1934) and others. As the asci mature, they



FIG. 222.—Apothecia of *Sclerotinia sclerotiorum* in the act of discharging ascospores by the puffing process. Note the white cloud of spores against the black background. (After Dickson and Fisher.)

imbibe water and develop considerable internal pressure. The ascus wall near the apex becomes soft and stretches until it suddenly gives way, releasing the pressure which forces the ascospores and the surrounding liquid out into the air. The liberation usually occurs as a series of sudden discharges, a phenomenon known as "puffing." The ascospores are forcibly ejected into the air for several centimeters, forming a white cloud of spores which may be carried off by air currents (Fig. 222). This process is usually interpreted as an adaptation to wind dissemination although the spores may not be disseminated exclusively by wind. The sudden discharge of spores may be set off in several different ways. Falck (1916, 1923) has classified

the discomycetes into two groups on the basis of the stimulus required for puffing. One group discharges its spores when warmed by radiant heat as from the sun; the other group responds to a slight touch of the fruiting body such as would be supplied by an insect alighting upon it. An insect alighting upon a ripe fruiting body would, by providing the stimulus for the spore discharge, become thoroughly contaminated with spores. It appears as if the puffing mechanism may not exclude the possibility of effective insect dissemination.

A striking example of adaptation of a fungus for insect dissemination is found in the anther smut of pinks [*Ustilago violaceae* (P.) Fckl.] as described by Brefeld and Falck (1905). This smut affects several species of plant of the pink family. Infection is accomplished through the flowers by spores that germinate on the stigmatic surface. The fungus is systemic and invades the entire plant, but spores are formed only in the anthers where they replace the pollen grains. The dark-colored echinulate smut spores are approximately the same size as the pollen grains; and, although they may be wind-disseminated, they are as readily transported by insects. The flowers are commonly pollinated by insects, especially by certain species of sphinx moths. The moths, in visiting flowers on infected plants, become contaminated with smut spores along with the pollen grains and transport them to the stigmas of healthy plants where they germinate and infect. Thus, by its habit of sporulating in the anthers, the fungus has become adapted to the more certain method of dissemination by the same insects that pollinate the flowers of its host plant.

A somewhat similar adaptation was described by Silow (1933) in a systemic disease of red clover caused by *Botrytis anthophila* Bond. The spores of this pathogen are disseminated by the bees that pollinate red clover. The spores, along with the pollen grains, are deposited on the stigma where they germinate and infect. The mycelium of the fungus grows into the developing seed and persists under the seed coat without affecting the viability of the seed. When the infected seeds germinate, they give rise to systemically infected plants. The fungus grows out into the young flowers, many of which are blighted; and the spores are formed over the surface of the shriveled anthers (Fig. 125). Bees on visiting the blighted blossoms in search of nectar become

contaminated with spores and in the continued search for nectar spread the infection to other plants.

Protozoa.—A few protozoa are recognized as plant pathogens. The methods of dissemination of endophytic protozoa have not been extensively investigated, but all available evidence points to insect transmission as the general rule. DuPorte (1924) mentioned two laticiferous plants (*Asclepias syrica* and *Convolvulus sepium*) in which the protozoa were seed-transmitted. No spores suitable for wind dissemination are produced by the protozoa. The endophytic protozoa appear to grow and multiply within the body of certain insects (Franca 1920 α) and to this extent may be considered as well adapted to insect dissemination. In fact, the nature of the organisms would make it difficult to account for their dissemination in any other way. When found in plant tissues, they occur only as intracellular parasites in the living latex cells. Since the latex cells are not exposed to the surface of the plants, it would be difficult to conceive of any other way in which the protozoa could gain entrance. In all probability, it will be found that practically all these endophytic protozoa are disseminated exclusively by insects. For a more complete discussion of insects and the endophytic protozoa see Chap. X.

Viruses.—The exact nature of the inoculum of viruses is not known. Whatever the inoculum may be, it exists in extremely small particles suspended in the protoplasm of the cell. That it is well suited for insect dissemination is evident from the fact that so many viruses are insect-transmitted. Many are transmitted only by insects so far as we know. It has been shown that infection by viruses depends upon the virus particles coming in contact with the living protoplasm in the cells or with the plasmodesmid strands connecting the cells. The sucking insects are ideal agents for introducing the virus into the living cells with the minimum exposure of the virus to unfavorable conditions.

Certain viruses are capable of surviving for long periods in the body of the insect vectors, and there is some evidence that a few of the viruses may actually increase there. These properties of the virus particles all add to the effectiveness with which they may be disseminated by insects. The extent to which the inoculum of viruses is capable of resisting desiccation, oxidation, and other unfavorable factors of the environment varies widely

and greatly influences their normal means of transmission. The virus of tobacco mosaic, for example, is very resistant to desiccation and may survive for many years in dried leaves, while the virus of cucumber mosaic will survive only a few hours of dryness. In general, those viruses which are unable to survive for very long *in vitro* are more dependent upon insects for transmission.

Seed Plants.—There are a few well-recognized plant pathogens among the seed plants. The common field dodder (*Cuscuta spp.*) and the mistletoes (*Loranthaceae*) are among the better known groups. The seeds formed by these pathogens are too large to be disseminated by insects, but the seeds of the mistletoes are especially adapted to dissemination by birds.

The seeds of the mistletoes are borne in a viscous and very sticky liquid that causes them to adhere tenaciously to anything with which they come in contact. The seeds adhere to the branches of the tree and germinate during rainy weather, sending haustoria into the xylem elements and absorbing water and mineral salts.

In the dwarf mistletoe (*Razoumofskyia spp.*), the seeds are forcibly ejected from the berry and are disseminated locally in this way. As the berry matures, it develops considerable internal pressure until a slight disturbance causes it to explode and eject the seed. The seeds may travel as far as 33 feet in a horizontal direction as a result of this force. For dissemination over longer distances, they are dependent upon birds which eat the berries and regurgitate the seed or pass them out in the excrement. The sticky seed also may adhere to the bird's beak later to be wiped off upon a branch of a tree. The seed may be distributed for relatively long distances by birds, and many of them so distributed are deposited in places favorable for infection.

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CHAPTER XV

THE FEEDING AND BREEDING HABITS OF INSECTS IN RELATION TO THE TRANSMISSION OF PLANT DISEASES

Insects constitute the largest of all animal groups, it having been estimated that approximately 75 per cent of all the animals of the world are insects. Nearly 700,000 species have been described and named, and it has been stated that there are probably several million species still undescribed. One finds an extremely wide variation in the life habits of such a large group of organisms. Even among the relatively small number of insects known to be concerned in the spread and development of plant diseases, we find such a variety of types in respect to their mode of life that very few generalizations can be made concerning their ability to transmit plant diseases. If we are to acquire a satisfactory knowledge of the general principles involved in insect transmission of plant diseases, it is desirable that we make a brief survey of some of the life habits of insects as they concern effectiveness in plant disease transmission. Inasmuch as the acts associated with feeding and breeding of insects are the ones most frequently involved in disease transmission, particular attention will be given to these activities.

THE FEEDING HABITS OF INSECTS AS RELATED TO DISEASE TRANSMISSION

In order to be an effective vector of a plant disease, an insect must visit, with a reasonable degree of regularity, both diseased and healthy plants. The mere association of an insect with diseased plants or the presence of inoculum on or in the insect's body is not sufficient justification for concluding that the insect is a vector of the disease. Unless the insect bearing the inoculum visits a healthy plant under conditions conducive to inoculation and infection, it may be of no significance in the transmission of the disease. Failure to recognize this fundamental principle

has been the source of frequent error in the study of insects in relation to the spread of plant diseases. This was the chief basis of the criticism made by Craighead (1916) of the work of Studhalter and Ruggles (1915) dealing with insects as vectors of chestnut blight. These authors demonstrated that a large number of insects collected from blight cankers were contaminated with spores of the blight pathogen and concluded that "they are important agents in the local dissemination of this disease." Craighead objected to this conclusion on the logical basis that most of the insects included in the study rarely visited or fed upon healthy trees.

One must be cautious, however, in concluding that an insect which apparently is associated only with diseased plants does not visit or attack healthy plants; for if the insect is a consistent vector of the disease and the plant always becomes infected following the insect attack, the situation easily may be misinterpreted. For example, it has been shown in work on potato blackleg and other bacterial soft rots that certain Dipterous insects, generally referred to as scavengers, are, in reality, responsible for initiating the decayed condition that is supposed to have attracted them. A careful study of the habits of the insect throughout its life cycle must be made before it can be disregarded as a possible vector.

It is generally recognized that ergot of cereals is transmitted by insects that feed upon the sugary exudate in which the conidia of the fungus are borne. A very large number of different kinds of insect have been observed feeding on the exudate, but they are not all equally effective in transmitting the disease. No accurate rating of the relative importance of these insects has ever been made, but it is known that several species of flies that are among the most common visitors to infected heads not only consume the exudate but feed also on the pollen of healthy heads. These pollen-eating species would naturally be more effective vectors than those species which are attracted to the heads of rye by the fungus exudate and which visit healthy heads only incidentally.

Another instructive example of the way in which the feeding habits of insects may influence the effectiveness of insects as vectors is furnished by the bark beetles that transmit the Dutch elm disease. These insects, principally *Scolytus scolytus* and

S. multistriatus, breed by preference in weakened or dying trees; rarely are they able to establish themselves in healthy vigorous trees. If it were not for the fact that the beetles feed for a period after emergence on the twigs of healthy vigorous trees, during which time they inoculate them with the pathogen, the disease would in all probability remain a relatively unimportant disease of unthrifty trees. But because of this feeding habit vigorously growing trees are inoculated and the disease becomes extremely destructive on elms regardless of their vigor. The native American bark beetle (*Hylurgopinus rufipes*) breeds also in infected elms and occasionally transmits the disease, but it is much less effective than the other bark beetles because it does not have the habit of feeding on the succulent twigs. However, according to Kaston and Riggs (1938), this species sometimes feeds upon the inner bark of the trees before breeding tunnels are established. The significance of this type of feeding in transmission of the disease has not been determined accurately, but there is reason to believe that it is of considerable importance (Kaston 1939).

It is of interest to note that the pine bark beetles (*Ips* spp. and *Dendroctonus* spp.) transmit blue-stain fungi that are capable of killing vigorous trees when artificially inoculated into them; but because the insects infest only weakened trees, the disease is largely confined to trees previously injured by some other cause. These beetles, unlike the *Scolytus* elm beetles, do not feed on the twigs. Transmission is accomplished entirely through the breeding tunnels.

The Mechanics of Feeding.—The mechanical process of feeding is a factor of prime importance in determining the effectiveness of an insect vector of a plant disease. Many insects that are frequent visitors to plants feed upon plant products but do not wound the plant in any way. As a general rule, these insects are not so effective as those which make feeding wounds, and yet some of them are important vectors. These usually transport the inoculum to blossoms or to wounds made by other agencies or else transport inoculum that does not require wounds for infection. The blossom-visiting insects are the most effective vectors of this class. As examples may be mentioned bees, wasps, and flies as vectors of fire blight, flies as vectors of ergot, and the sphinx moths as vectors of anther smut of pinks. Insects that transport inoculum to wounds made by other

agencies are, for the most part, incidental vectors and of minor importance. In such cases, wind or other agent of dissemination is usually of greater significance than insects although there are some important exceptions.

Those insects which make feeding wounds are, by far, the more effective vectors of plant diseases. Many different kinds of wounds are made by insects in the process of feeding, and the type of wound is a factor of much significance in determining infection. No two species of insect feed in exactly the same way, and very minor differences may have a great influence on the effectiveness of the insect as a vector.

Some of the more common types of feeding wound are those made by the chewing insects. These may consist of a simple crushing of plant tissue by such insects as grasshoppers, caterpillars, and many beetles; or they may take the form of burrows extending deep into the tissues, such as those made by wood-boring beetles. It would be difficult to make any generalizations as to which of these two kinds of injury is more conducive to insect transmission; much depends upon the nature of the disease being transmitted. In general, the feeding wounds made by such chewing insects as grasshoppers and potato bugs, because of exposure to the air, dry quickly and are relatively unimportant as points of infection. On the other hand, similar wounds made by the cucumber beetles on the stems of cucurbits are effective avenues of entrance for the vascular bacterial parasite *Erwinia tracheiphila*.

Insects that burrow deep into plant tissues provide wounds that often are ideally suited for infection, although there are many exceptions. When the adult insects burrow into the plant, as do the bark beetles, many fungus spores are introduced and the tunnels provide ideal moist chambers for growth of the fungus and for infection. Other insects burrow in as young larvae, freshly hatched from eggs, and having had less opportunity for becoming contaminated are less likely to introduce inoculum. This difference may be illustrated by comparing the bark beetles that burrow in pine logs as adults and introduce large quantities of spores of the blue-stain fungi with the wood-boring *Monochamus* spp. and the buprestid beetles which bore into the same logs as freshly hatched larvae and introduce few, if any, spores of pathogenic fungi.

Chewing insects play a relatively unimportant part in the transmission of virus diseases. This can be attributed largely to the type of wounds they make. The cells bordering the wounds made by chewing insects are usually crushed and killed so that a virus introduced into the wound would rarely come into contact with living protoplasm, a recognized requirement for infection by all viruses. Insects of this type are capable of transmitting only those viruses which are very highly infectious, being readily transmitted by artificial sap inoculation.

The rasping-sucking type of feeding, represented by that of the larvae of Dipterous insects, results in wounds well adapted to transmission of certain kinds of diseases. This type of wound is usually made in succulent tissues favorable to infection by bacteria and fungi, and the larvae burrow into the tissues to avoid the injurious effects of light and desiccation. The continuous nature of the wound tends to counteract the wound-healing properties of the plant tissues and affords an excellent opportunity for successful inoculation.

The piercing-sucking insects make wounds that are ideally adapted to the transmission of plant diseases. They penetrate deep into the tissues with their mouth parts, and any pathogen introduced into such a wound is not exposed to any of the ill effects of light, desiccation, or oxidation. When the pathogen is transmitted biologically by the insect, the secretion of saliva into the wound provides a means of introducing the required quantity of inoculum. It would, however, be unwise to generalize to any great extent as to the significance of the injection of saliva; for, as has been shown by Smith (1920), the capsid bugs are not capable of transmitting many of the plant viruses. The saliva injected into the plant by these insects is highly toxic to plant cells and kills much of the tissue immediately surrounding the puncture. Smith believes that the rapid killing of this adjacent tissue prevents the viruses from becoming established in the plant and is responsible for the failure of this particular group of sucking insects to transmit viruses effectively. This conclusion is supported by the generally recognized fact that the viruses behave as obligate parasites and are not able to multiply except in direct contact with living tissue. It is also possible that the saliva of some insects may be toxic to some kinds of inoculum.

The mechanics of feeding by insects is of particular significance in the study of the transmission of the virus diseases. Brandes (1923), working with *Aphis maidis* and sugar-cane mosaic, was among the first to study the mechanics of feeding of insect vectors in relation to virus diseases, although as early as 1891

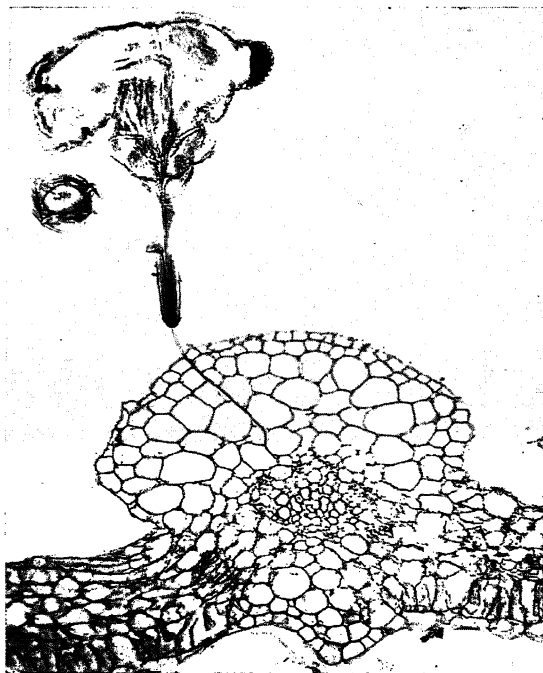


FIG. 223.—A section through the head and mouth parts of an aphid feeding upon the vein of a potato leaf. Note that the setae have located and penetrated the phloem tissues. (After Dykstra and Whitaker.)

Büsgen had studied the methods of feeding of aphids in connection with his investigation of the formation of honeydew. The feeding wounds of aphids (*Rhopalosiphum dianthi* Schrank) and the path followed by the setae were described also by Woods (1900) in his study of stigmonose of carnations. In 1923, Davidson described the penetration of plant tissues by *Aphis rumicis* L. In the same year, a similar study was reported

by Horsfall (1923) with respect to *A. rumicis* L., *Myzus persicae* Sulz., *Macrosiphum rubbeckiae* Fitch, and *M. ambrosiae* Thomas. This was followed by the more extensive comparative study of the feeding methods of several Hemiptera in particular relation to virus transmission as reported by Kenneth Smith (1926). The mechanism of feeding has been described for several leaf hoppers by Smith and Poos (1931) and by Smith (1933); and similar studies dealing with the beet leaf hopper [*Eutettix tenellus*

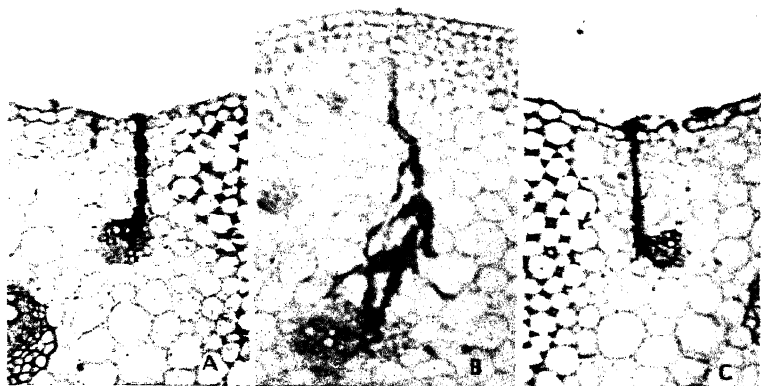


FIG. 224.—Three sections through petioles of sugar-beet leaves at the point of feeding punctures of *Eutettix tenellus*, showing the sheath of heavily staining material that surrounds the path of the setae. Note the change of direction and the results of probing to reach the phloem in B. (After Bennett by courtesy of the U. S. Department of Agriculture.)

(Baker)], with special reference to the transmission of curly top, were made by Bennett (1934).

These studies, along with other less extensive ones, have given us a fairly clear picture of the nature of the feeding punctures of the most important groups of vectors of virus diseases. In general, those Homopterous insects which are most effective in transmitting viruses feed upon the phloem (Figs. 223 and 224). The stylets may penetrate the cell walls of the epidermis or may push in between epidermal cells. Penetration of the cortex parenchyma may be either intercellular or by direct penetration of the cell walls. As a rule, the setae of aphids follow an intercellular path although cell-wall penetration is often observed. The leaf hoppers, on the other hand, usually puncture a higher

percentage of the cortex cells in a more direct path to the phloem. The path followed appears to be determined largely by the size and stiffness of the setae and the relative thickness of the cell walls of the plant tissues fed upon.

In the feeding of both leaf hoppers and aphids, there is a copious flow of saliva into the intercellular spaces from the beginning of penetration. This secretion reacts with the plant sap and forms a sheath that completely surrounds the penetrating setae (Fig. 224). There has been a difference of opinion as to the nature of the sheath material. Horsfall (1923) interpreted it as of plant origin, being formed as a wound reaction. However, the opinion of most workers is that the sheath is largely of insect origin. Smith (1933) presented strong evidence from microchemical tests to support this view, and Bennett (1934) has observed the formation of a typical sheath of *E. tenellus* while feeding through a membrane on hanging drops of a synthetic solution.

The function of the setal sheath is not definitely known. It has been suggested that it serves to make an airtight seal about the setae, thus preventing the leakage of air or liquids along the sides and increasing the efficiency of the pumping mechanism used for sucking out the contents of the phloem. The sheath also may serve to protect the setae from any injurious action of the cell sap and may provide better lubrication for their movement. There is also some evidence that the salivary secretions aid the penetration of the smaller setae by dissolving the middle lamellae of the cells. In any event, the feeding mechanism is a very efficient one. Since it has been shown that the salivary secretion of many vectors is the medium in which the virus is introduced into the plant, a more perfect mechanism for inoculation scarcely could be imagined.

The mechanism of feeding is important, also, in the study of the nonparasitic diseases caused by toxicogenic insects. Diseases of this type (psyllid yellows, green spot of pineapple, hopperburn, etc.) have been attributed in general to diffusible toxic substances introduced in the saliva of the insect. However, this interpretation has been questioned in the case of hopperburn by Smith and Poos (1931), Smith (1933), and Johnson (1934), who maintain that the setal sheath, together with the mechanical injury to the plant tissues, results in a clogging of the phloem

vessels that interferes with the normal translocation of plant materials and accounts for pathological symptoms of hopperburn. There is need for further study of the mechanics of feeding and associated phenomena in interpreting the nature of the diseases caused by toxicogenic insects.

Food Plants.—The choice of food plants by insects has an important bearing on their ability to transmit diseases successfully. Among the first things to be determined in a study of insect transmission is the host range of the disease and range of food plants of the insect in question. Practically all plant diseases affect to some extent one or more species of noncultivated plants which may serve as reservoirs for the pathogen. Weeds and other noncultivated plants rank among the most common sources of infection for the cultivated crop. Also, many insect pests of crop plants feed upon wild plants in some stage of their development or during some part of the year. Outbreaks of many insect-transmitted plant diseases can be traced directly to insects that move from infected wild plants to the cultivated crop. The movement may be simple dispersion from breeding centers or definite directional migration. This factor is especially important in respect to virus diseases, as may be illustrated by the curly top of sugar beet and *Eutettix tenellus*, the mosaic of cucurbits and *Aphis gossypii*, and many others. Even though the noncultivated plants may not be susceptible to the disease in question, they may serve as breeding places for the vectors, and a knowledge of this fact is equally important in the control of insect-transmitted diseases.

Mader (1937) has reported that in New York the presence of yellow dwarf of potato transmitted by the clover leaf hopper (*Aceterogallia sanguinolenta*) is closely correlated with the presence of medium red clover (*Trifolium pratense*). Since the leaf hopper is abundant in regions where the disease is not prevalent, the clover, which is a suspect of the disease, probably serves as a source of viruliferous vectors that move from the clover to potatoes. If this relationship is sufficiently constant, a change in cropping practices may be more effective in controlling the disease than attempts at direct control of the vector.

Wellman (1935) has studied and charted the spread of southern celery mosaic by *A. gossypii* Glov. in relation to weeds as sources of infection. This virus affects many species of plants, including

weeds of various types, *Commelina nudiflora* L. being the most important of its weed hosts. This is a perennial plant in which the virus overwinters and on which the aphids feed, especially when more tender annual plants are not abundant. Careful studies have shown that infection in celery starts from infected weed patches along the edge of the field and spreads progressively through the field.

When the annual plants die or are chopped down, aphids leave and carry the virus back to nearby perennials, reestablishing themselves on old plants, or colonizing and infecting the young perennials, which act as reservoirs of the virus over periods unsuited to growth of the more tender plant species. It is upon advent of new growth of many of the annuals that dissemination of the disease becomes noticeable. The virus-carrying aphids migrate back from the less succulent and palatable reservoir hosts to annual crops and weeds, and the virus then becomes economically significant.

A considerable degree of control of celery yellows has been demonstrated by the thorough local eradication of *C. nudiflora* in the vicinity of celery fields.

Heinze and Profft (1938) made an extensive study of the life history and distribution of *Myzus persicae* Sulz. in Germany in relation to the prevalence of virus diseases of potatoes. It was concluded that this aphid alone is responsible for the transmission of the three most important potato viruses in Germany (leaf roll, γ and α virus) and that there is a close correlation between its abundance and the difficulty of producing virus-free seed stock. It was shown furthermore that the abundance of the aphid in various parts of Germany is largely determined by the abundance of peach trees. *Myzus persicae* is a polyphagous aphid, but it utilizes the peach exclusively as the winter host. In early fall, the winged form of the insect appears and migrates from potatoes (and its many other host plants) to the peach trees on which the sexual generation develops. Eggs are deposited on the peach trees in late October. They lie dormant until early spring, then hatch, giving rise to wingless females that reproduce viviparously. About 2 months later (approximately May 15 in Berlin), the winged individuals appear. Within a few days, these begin to migrate from the peach trees to potatoes and other summer host plants. Since the virus is not congenitally transmitted in the aphids from one generation to the next, the virus

must be acquired from infected potatoes or from systemically infected weeds.

Extensive aphid counts in potato fields throughout the season in various sections of Germany showed clearly that *M. persicae* was much more abundant in those regions where peach trees were more numerous and that these regions coincided with those previously recognized as unfavorable for the production of virus-free potatoes. On the basis of these facts, it was recommended that seed-potato production be limited to those regions where peach trees are not economically important and that, in regions devoted to intensive seed production, as many peach trees as possible be eliminated.

Murphy and Loughnane (1937), in a 10-year study of the spread of leaf roll in Ireland, state that *M. persicae* winters in an active state on cabbage and that the relative freedom of potatoes from leaf roll in eastern Ireland is probably due to the absence of winter hosts of the aphids near the potato fields.

Palmer (1938) has called attention to the importance of knowing the host range and relationships of aphids in respect to their role as vectors of virus diseases. Aphids are grouped into the following three classes on the basis of their host relationships:

Single-host aphids are aphids that feed upon only one species of plant or on a few closely related species of a single genus. *A. pomi* DeG. on apple and *Macrosiphum rosae* (L.) on rose are examples of this class.

Polyphagous aphids infect a relatively large number of plant species in several distantly related families. Examples are *A. gossypii* Glov. and *A. medicaginis* Koch, each of which feeds on plants in 11 different families. *Aphis rumicis* L. and *M. persicae* Sulz. each feeds on plants in eight families. These plants include both weeds and cultivated crop plants. Vectors of virus diseases with polyphagous habits add greatly to the complexity of virus disease control.

Alternate-host aphids commonly feed on a winter host, usually woody, and one or more herbaceous summer hosts. For example, *M. persicae* utilizes the peach as a winter host and feeds on many herbaceous species in summer. *Aphis bakeri* Cowen winters on apple and feeds on clover in summer.

The time of migration is often of great significance in the study of the relation of aphids to the spread and development

of virus diseases. *Alternate-host migrants* are those winged aphids which leave the first host to migrate to the alternate one. The migration usually occurs in late spring, but the exact date varies with the species and with the climate. *Single-host migrants* are the winged forms of the single-host aphids that are responsible for the dispersion of the species on its preferred host plant. The single-host migrants appear some time in late spring.

It is well known that some aphids transmit only one plant virus whereas other species transmit several different viruses. In general, it is the polyphagous aphids such as *M. persicae* that transmit more than one virus. The single-host aphids as a rule transmit only a single virus.

The plants upon which toxicogenic insects have fed may determine their ability to produce a disease as shown by Carter (1936b) in his study of the green-spotting disease of pineapple caused by the pineapple mealy bug [*Pseudococcus brevipes* (Ckl.)]. When the insects were removed from *Panicum* grass to pineapple, they failed to cause the greenspotting; but, when taken from any other host, they readily caused the disease.

The feeding habits of insects and their choice of host plants are often influenced by cultural practices with striking effects on their importance as vectors of plant disease. Thus, *A. maidis*, which is considered to be the principal vector of sugar-cane mosaic, does not normally feed upon sugar cane but develops abundantly on certain wild grasses that are preferred food plants. In the tropics, these wild grasses must be cut down periodically as a cultural necessity. When this is done, the vector migrates to sugar cane and in so doing transmits the virus from the wild grasses. In subtropical regions, the wild grasses die in winter, and then *A. maidis* feeds upon sugar cane until a new supply of grasses is available. Ingram and Summers (1936) claim that in Louisiana the rusty plum aphid [*Hysteroneura setariae* (Thomas)] is the principal vector of sugar-cane mosaic although it does not transmit the virus so readily as *A. maidis*. The sugar cane plant, however, is a preferred food plant of *H. setariae*, which is much more numerous on sugar cane and is there for a longer period of time.

Insect Motility.—The motility of insects and their migration habits are of considerable importance. Some insects, as the beet leaf hopper, have definite periods of migration in which wide areas of cultivated plants may become infested in a short period of

time. Other insect vectors spread slowly from centers of infestation and the diseases transmitted by them may be correspondingly slow in their spread. Aphids in certain stages of development are relatively inactive and move very slowly from plant to plant. The rate of spread of a virus disease by aphids may be increased by any cultivation practice that increases their motility. The practice of roguing virus-infected plants from a field may defeat its own purpose if the vectors feeding upon the diseased plants are allowed to move to the remaining plants. Incomplete control of insect vectors by spraying or dusting may likewise hasten the spread of the disease by dispersing viruliferous insects from diseased to healthy plants.

The prevailing winds or strong winds of some duration may influence greatly the incidence of a disease by its influence on the movement of the insect vectors. Wellman (1935) has shown that the spread of celery mosaic in Florida is dependent upon the movement of the aphid vector (*Aphis gossypii*) and that this is influenced greatly by the direction of the prevailing winds. The winged forms of the vector may fly for a considerable distance and are responsible for new centers of infection, but their direction of flight is determined largely by the direction of the prevailing wind.

Felt (1937) has studied the influence of the prevailing winds on the movement of the elm beetle (*Scolytus multistriatus* Marsh.) and its significance in the spread of the Dutch elm disease in the United States. The study of wind drift by balloons in the regions infected with the Dutch elm disease indicates a drift in a northeasterly direction, which coincides very closely with the direction of greatest spread from the early centers of infection. Although no studies with the beetles have been made, there are several records of bark beetles being caught high in the air in airplane traps. Felt is of the opinion that "wind carriage of infected beetles affords the most reasonable explanation of the distribution of the Dutch elm disease in the northeast" and that balloon distribution data will indicate the general lines of further spread. Fransen (1939) has observed that the elm bark beetles fly high above the treetops and that the taller trees are most frequently attacked whereas the lower ones escape.

The migration habits of insect vectors should be studied more extensively than they have been in the past. Many vectors of

plant diseases feed for a time on certain species of wild plant, later migrating to the cultivated crops. The causes of migration may vary with different vectors. The beet leaf hopper apparently migrates from the native vegetation to the sugar-beet fields when the native vegetation dries up or matures with the approach of the hot, dry summer weather. Carter (1930) has shown, however, that the leaf hoppers migrate not alone because of the scarcity of food, but because they have an inherent instinct to migrate. This highly developed instinct probably evolved by natural selection in heterozygous populations, through the greater survival of those individuals with migrating instincts, as the native vegetation dried up year after year. The variability of the migration instinct and the time required for a change in migration habits are not known. If appreciable changes can be effected in a relatively few generations, the migration and food habits of insects could be modified strikingly by climatic cycles or by the changing agricultural practices of a region. Such changes would be of considerable significance in epiphytology and might explain the great fluctuation often observed in the prevalence of insect-transmitted diseases.

Insects that are active and move from plant to plant frequently are more effective in transmitting viruses than the more sedentary species, other things being equal. This is a factor that should be kept in mind when evaluating the relative importance of different vectors. A very active species that is capable of transmitting a virus in only a small percentage of opportunities may be of greater importance than a more sedentary species that transmits the disease in a higher percentage of opportunities. Greenhouse experiments demonstrating that a given insect is capable of transmitting a virus should be supplemented by field studies of the habits of the insect before its role as a vector is finally evaluated.

Tissue Selection.—Many insects that feed upon a given plant do not do so indiscriminately. Nearly all species have a particular preference for certain tissues, and some are strictly limited in respect to the tissues on which they can feed successfully. This fact has a very important bearing on the subject of insect transmission of plant diseases. Slight differences in feeding habits and choice of tissues are often not noticed, and the significance of these differences is frequently overlooked until

revealed by careful and detailed study. This aspect of the problem of insect transmission was greatly neglected for many years and still is in need of more careful study.

The importance of tissue selection is strikingly illustrated by the investigations of Bennett (1934, 1937), Bennett and Esau (1936), and Fife and Frampton (1936). These workers have shown that the virus of curly top of beets is confined almost entirely to the phloem tissues of affected plants. The virus is rarely found in the parenchyma tissue. It was demonstrated also that the beet leaf hopper normally feeds on the phloem and that when viruliferous leaf hoppers feed only on tissues other than phloem they rarely, if ever, inoculate the plant with the virus. There is, therefore, a close correlation between the tissue selection by the leaf hopper and its effectiveness as a vector of curly top.

Fife and Frampton (1936) have offered the first plausible explanation of the method by which the leaf hopper is able to locate and select the phloem on which to feed. This explanation is based on the existence of a pH gradient between the cell contents of the phloem and that of the parenchyma. By using a potentiometer and a quinhydrone micro-electrode to measure the acidity of the cell sap in various tissues, it was found that the sap of the phloem is considerably more alkaline than that of the surrounding parenchyma, resulting in a pH gradient as illustrated graphically in Fig. 146. It was shown also that the reaction of the saliva of the leaf hopper is alkaline, approximating that of the liquids of the phloem. These facts led to the hypothesis that the pH gradient was utilized by the leaf hoppers as a guide to the phloem. According to this hypothesis, when the setae of the insect penetrate the parenchyma, the difference in acidity between the cell sap and the insect's saliva gives rise to an undesirable reaction, which causes the leaf hopper to withdraw or change the direction of its setae. The process is continued with the result that the pH gradient leads to the phloem where the desired alkalinity is found.

This hypothesis was supported by some additional experimental evidence. Leaf hoppers were placed in artificial feeding cages where, by piercing a thin membrane, they could feed on drops of sugar solutions adjusted to two different pH values (pH 8.5 and pH 5.0). They were observed to explore the acid

drops with their setae and move on to the alkaline ones before finally settling down to feed.

The pH gradient normally found in beet-leaf tissue was modified by subjecting the beets to high concentrations of carbon dioxide. This treatment tended to equalize the pH values of the phloem and parenchyma as shown in Fig. 147. When leaf hoppers were fed on these treated beets, they were no longer able to locate the phloem and fed indiscriminately on different tissues with a corresponding decrease in effective inoculation with the virus. These studies constitute an important contribution to



FIG. 225.—Lesions made on alfalfa leaves by two species of *Empoasca* which do not select the phloem on which to feed. A, a leaf fed upon by one adult of *Empoasca abrupta*; B, by one adult of *E. erigeron*. Approx. $3\frac{1}{2}\times$. The stippling effect is much less severe than the burning effect of *E. fabae*. (After Smith and Poos.)

the study of insect transmission of viruses, and similar studies should be made with other viruses and their vectors. A further study of tissue selection by insects along with similar studies of the localization of the viruses in plant tissues may explain many cases of apparent specificity between certain virus diseases and their insect vectors.

There is some evidence that the ability of aphids to transmit leaf roll of potato is associated with phloem feeding (Dykstra and Whitaker 1938). This virus is transmitted by several species of aphid, but those which consistently feed upon the phloem (*Myzus persicae*, *M. circumflexus*, and *M. solani*) transmit it more readily than *Macrosiphum solanifolii* which feeds more frequently on mesophyll tissues. On the other hand, the three

species of *Myzus* varied in their ability to transmit certain viruses of the potato although no differences in their feeding habits were observed.

Tissue selection by piercing-sucking insects is significant, also, in the study of toxicogenic insects and the diseases caused by them. Smith and Poos (1931) have studied the tissue selection of six species of *Empoasca*. Five species (*E. maligna*, *E. abrupta*, *E. filamenta*, *E. bifurcata*, and *E. erigeron*) selected the mesophyll tissue of the more mature leaves upon which to feed. They caused only slight injury characterized by a spotting or stippling of the upper leaf surface (Fig. 225). In contrast, *E. fabae* showed a decided preference for the phloem tissue. In feeding on the phloem, this species seriously disrupts the normal functions of the vascular elements, and the destructive pathological condition known as hopperburn then results.

THE BREEDING HABITS OF INSECTS AS RELATED TO DISEASE TRANSMISSION

The effectiveness of an insect as a vector of a plant disease often may be influenced by its habits of breeding. Plant wounds made by insects in connection with breeding activities frequently serve as points of entrance for plant pathogens. The adults of some species tunnel into plant tissue and establish brood tunnels in which the eggs are deposited and the young are reared. The ambrosia beetles and the bark beetles breed in this way. Various kinds of fungi are usually associated with insects of this type, and in many cases the fungi are pathogenic to the plants in which the insects breed, e.g., *Cerostomella ips*, *Tuberculariella ips* causing the blue stain of conifers, and *C. ulmi* causing the Dutch elm disease.

The females of certain other species of insect are equipped with sharp-pointed ovipositors with which they make punctures in plant tissue for the insertion of their eggs. In general, this type of oviposition wound is not so important in the transmission of plant diseases as the one just described, but there are a few noteworthy examples of effective transmission through oviposition. The female apple-maggot fly (*Rhagoletis pomonella* Walsh) inserts her eggs in punctures made with a sharp ovipositor in the tissues of apple fruit (Fig. 93). Pathogenic bacteria (*Phytophthora melophthora* Allen and Riker) are introduced along with the eggs

and eventually cause a decay of the apple (Allen, Pinckard, and Riker, 1934). The olive fly (*Dacus oleae* Rossi) is said to inoculate olive twigs with the olive-knot pathogen [*Erwinia savastanoi* (Smith) Bergey *et al.*] in a similar manner (Petri 1910) (Fig. 90).

A unique example of inoculation at the time of oviposition is that of tree crickets and the canker of apples caused by *Leptosphaeria coniothyrium* (Fckl.) Sacc. as described by Gloyer and Fulton (1916). The female tree crickets insert their eggs into punctures made with their ovipositors in the apple twigs. The punctures are then plugged with materials which often contain spores of the pathogen. Two species (*Oecanthus niveus* DeG. and *O. exclamationis* Davis) plug the puncture with pellets of chewed bark often obtained from cankers on which the fungus is fruiting. Other species use pellets of feces which also commonly contain viable spores of the fungus. The peculiar instincts manifested by these insects during oviposition are thus responsible for their importance as vectors of apple canker (Fig. 128).

Many insects deposit their eggs on the surface of plant tissue without making wounds, but when the eggs hatch the larvae often bore into the tissues and inoculate them with plant pathogens. The part of the plant on which the eggs are deposited may be significant in determining the amount of damage caused by the disease. For example, the Dipterous insect vectors (*Scaptomyza graminum* Fall. and *Elachiptera costata* Leow) of celery heart rot, caused by *Erwinia carotovora*, deposit their eggs on the surface of the celery leaves. In rainy weather, the eggs are deposited on the outer leaves, and the resulting soft rot is not very destructive. In dry weather, on the other hand, the insects seek a moist place to deposit their eggs and lay them on the young heart leaves near the growing point. When these eggs hatch and the larvae inoculate the heart leaves with the bacteria, the growing point is killed and much loss follows (Leach 1927). The instinct that leads the insect to seek a moist place for depositing its eggs obviously greatly influences its significance as a vector.

Most insects have decided preferences among the host plants on which they breed. Insect vectors often breed more abundantly on weeds and wild plants than they do on the economic plants on which they feed and to which they transmit diseases. The control of many insect-transmitted diseases depends upon the destruction of the primary breeding hosts of the vector. For this reason, no study of an insect vector is complete until its

breeding habits, including its preference of breeding hosts, are accurately known. It has been determined that the plant bugs which transmit stigmatomycosis to cotton and other plants breed extensively on many weeds and noncultivated plants. Control measures for the disease depend largely upon keeping down the population of vectors through destruction of their weed hosts.

The breeding habits of insects in relation to cultural practices are also important from this standpoint. In the control of yellow top of alfalfa caused by the leaf hopper (*Empoasca fabae* Harris) it has been shown (Graber and Sprague 1933, 1935; Searls 1934; and Jewett 1934) that the population of hoppers and the subsequent injury can be greatly reduced by timing the first cutting to coincide with the end of the heaviest egg-laying period but to precede the hatching period. By doing this, the majority of the eggs of the first brood of hoppers is destroyed when the alfalfa is cut, and the second brood is so greatly diminished that it fails to cause much damage. The success of this method of control is greatly influenced by the weather conditions and the relationship between the stage of development of the insect and the growth stage of the alfalfa.

Insect Abundance.—If a plant disease is largely dependent upon insects for its transmission, it is only reasonable to suppose that the prevalence of the disease will be influenced by the prevalence of its insect vector. This would be especially true of such diseases as bacterial wilt of cucurbits and certain virus diseases that are transmitted only by certain species of insect. A few observations have been recorded which indicate that such relationships exist. The influence would be less on diseases in which insects constitute only one of several agents of inoculation. It is a matter of common observation that the prevalence of both insects and plant diseases varies from year to year, but remarkably few studies have been reported in which the influence of fluctuating insect populations on the fluctuating prevalence of diseases has been considered. It is recognized that the populations of certain insect species fluctuate in definite cycles as a result of parasitism and other biotic factors. As the insect pest becomes more abundant, its parasites also may multiply. Soon the parasites kill so many of the host insects that the latter become relatively scarce. The scarcity of the host insect results in a diminishing of the population of parasites until the host insect again becomes more abundant and the cycle is repeated.

Cycles of this type have been recorded for a number of insect vectors, and there are numerous records of correlation between the prevalence of a vector and the disease that it transmits. Plant pathologists, seeking to explain the variation in the prevalence of an insect-transmitted disease, should endeavor to learn as much as possible about the fluctuations in population of the insect vector and their possible relation to the fluctuations in prevalence of the disease.

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CHAPTER XVI

INSECT TRANSMISSION OF ANIMAL DISEASES COMPARED WITH INSECT TRANSMISSION OF PLANT DISEASES

The sciences of plant and animal pathology have much in common. The phenomena dealt with are essentially the same, although the two sciences are cultivated by different groups of workers, each highly specialized in its own field. Each group is concerned with the fundamental biological processes of disease, *i.e.*, parasitism, pathogenicity, inoculation, infection, epidemiology, immunity, etc. A discovery made in one field often has direct application in the other and still more often may be the key to the solution of some vexing problem. Partly because of the higher values placed upon human lives, or the lives of animals, animal pathology developed somewhat earlier and has progressed more rapidly than plant pathology. Consequently, the younger science of plant pathology has in the past drawn much from the rich discoveries of medical science and may be expected to do so in the future. Therefore, a student interested in the subject of insects in relation to plant diseases will do well to acquaint himself with the known facts concerning the relation of insects to the spread and development of animal diseases.

An exhaustive treatment of the subject of insects in relation to animal diseases is beyond the scope of this book. It will be sufficient here to give a brief historical account of the development of medical entomology and, for the purpose of comparison, to give the essential facts concerning a few representative examples of insect transmission of animal diseases. For further information on the subject, the reader is referred to the well-known works of Matheson (1932), Riley and Johannsen (1932), Fox (1925), and Wenyon (1926) and the literature cited by them.

HISTORICAL

Interest in insects from the standpoint of their relation to the transmission of animal diseases is a relatively recent develop-

ment. The first general treatment of the subject was published in 1899 by Nuttall, who stated the situation at that time as follows: "Very few of the works on hygiene even mention the role of insects as carriers of infection, and those that do generally speak vaguely on the subject." But since this statement was made, there has been a rapidly increasing interest in this aspect of entomology and medicine; and as new facts have been discovered, insects have proved to be a factor of prime importance in medical science. The past 40 years has witnessed the rapid development of the broad but specialized field of medical entomology.

Despite what has been said, the idea of insect transmission of disease is not of recent origin. Various "theories" of insect transmission of human diseases date from the ancients. Experimental proof, however, was lacking; and the theories failed to influence scientific thought. With the first experimental proof of insect transmission, the theory became a fact, and the idea was rapidly exploited, stimulating further research in the field. With the exception of early inconclusive experiments dealing with insects and anthrax, the first experimental evidence that insects are concerned in the transmission of diseases is that published by Manson (1878, 1884) showing that mosquitoes are vectors of the filarial worm causing elephantiasis of man. Manson showed that the larvae of the parasite are taken up in large numbers by the mosquito when it sucks the blood of an infected person. He showed that the worm undergoes cyclic development in the body of the mosquito. Manson thought that the parasite was released in the water on the death of the mosquito and that man became infected by drinking contaminated water, but he was in error on this point. The true method of infection was discovered in 1900 by Low, who showed that infection followed the bite of an infected mosquito. The incompleteness of the work of Manson does not detract much from the significance of his contribution, for it influenced greatly the work of others and marked the beginning of medical entomology.

The next great step forward was made by Smith and Kilbourne with their classic work on the cattle tick and the Texas fever of cattle, published in 1893. The tick, shown to be a vector of Texas fever, is not a true insect but a closely related arthropod. This work is of special historical significance because it marks the first proof of the transmission of a protozoal disease

by a bloodsucking arthropod. The recognition of this relationship and its significance in the epidemiology of animal diseases led to the discovery later of the vectors of the Rocky Mountain spotted fever of man, tularemia, and other similar diseases.

A few years later (1895) Bruce, working in Africa, discovered that the fatal nagana disease of cattle is caused by a blood-inhabiting trypanosome and showed that the disease is conveyed from sick to healthy animals by the bloodsucking tsetse fly (*Glossina morsitans*). This discovery paved the way for the demonstration of the cause and method of spread of the insidious and fatal African sleeping sickness of man, a disease very similar to the nagana disease of cattle. It is caused by *Trypanosoma gambiense* and is transmitted by several species of *Glossina*, or tsetse fly. Bruce and many other workers, including both entomologists and medical men, contributed to the solution of this problem.

The next great advance, and one of much significance, was the proof of the transmission of malaria by mosquitoes. On the basis of observational evidence, many laymen as well as scientists had expressed the idea that mosquitoes were concerned in the spread of malaria, but in the absence of experimental evidence the theory had received little credence. As early as 1880, Laveran had shown that malaria was caused by a parasitic protozoan and had suggested that it might be transmitted by mosquitoes. However, the major credit for experimental proof of the transmission belongs to Ross who, encouraged and inspired by Manson, worked out the development of the malarial parasite in the body of the mosquito. The publication of this work was soon followed by its confirmation and extension by many different investigators.

A great impetus to research in this field and the most dramatic of them all was the proof of the transmission of yellow fever by the mosquito. This proof was supplied by the United States Army Commission headed by Walter Reed (1900), who was assisted by James Carroll, Jesse Lazear, and Aristides Agramonte. The significance of this discovery in the control of yellow fever is too well known to need repetition here. It is sufficient to say that it made possible the rapid suppression of one of the most dreaded diseases of mankind against which medical science previously had been entirely helpless. But this was not all.

For once and all, it impressed upon the mind of scientist and laymen alike the importance of insects in the spread and development of diseases. Entomology became an essential part of medical science.

The last 35 years have seen a remarkable growth in the field of medical entomology. Insects have been shown to play a vital part in the epidemiology of a dozen or more diseases of major importance affecting man or animal. The story of the investigations leading to these discoveries is one of the most fascinating chapters in the history of medical science. For the romantic and human interest side of the story, the student should read the books of DeKruif ("The Microbe Hunters," 1926), Howard ("Yellow Jack," 1933), and Kelley ("Walter Reed and Yellow Fever," 1906).

EXAMPLES OF ANIMAL DISEASES TRANSMITTED BY INSECTS AND RELATED ARTEROPODS

In order that we may have a basis for comparing the relation of insects to the spread and development of man and animals with similar phenomena in plant pathology, it will be necessary to consider a few representative animal diseases that are transmitted by insects. They will be discussed primarily from the standpoint of insect transmission. For more complete accounts of other aspects of the diseases, the reader must consult the standard medical texts. The diseases will be considered also in the light of present-day knowledge, and it should be remembered that much information has been added since the work of the pioneers mentioned above.

Mosquitoes and Filariasis.—Filariasis is a disease of man caused by nematode worms that inhabit the blood and lymph. A variety of symptoms are caused, one of the most common being enormous swellings on various parts of the body, the condition being known as "elephantiasis." The swellings are often accompanied by ulcers and other disorders arising from the stoppage of the lymphatic trunks. The pathogen is a threadlike round worm (*Filaria bancrofti* Cobbold). The disease is indigenous in nearly all tropical and subtropical countries. In the United States, it occurs in a small area around Charleston, S. C.

Transmission of Filariasis.—Mosquitoes are the vectors of filariasis. Fifteen or more different species are known to transmit it, and no other method of spread in nature is known. The blood of man is the natural habitat of the filarial worm, and man is its definitive host, *i.e.*, the host in which the sexual stage of the parasite is found. Larvae occur in great abundance in the blood of affected individuals, as many as 600 being found in a single drop. The larvae, whose average size is about 8 by 300 microns, are sucked up with the blood by mosquitoes when they feed on

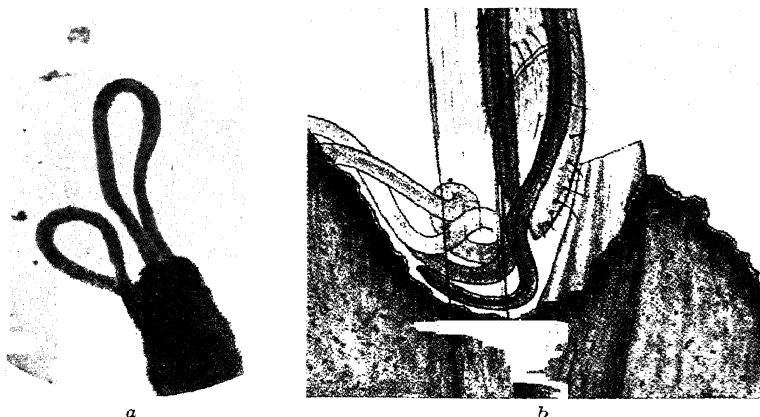


FIG. 226.—*Filaria* larvae emerging from the labium of a mosquito. *a*, larvae emerging from labium of *Aedes variagatus*; *b*, a semidiagrammatic sketch showing larvae leaving labium of a feeding mosquito and burrowing into the skin. (From Manson's "*Tropical Diseases*.")

an infected individual. The larvae enter the stomach of the mosquito, escape from their sheath, bore through the intestinal wall, and migrate into the thoracic muscles. Here, in the course of 16 to 25 days, they undergo metamorphosis but do not increase in number. The mature larvae then migrate into the labium of the mosquito. The larvae, which are too large to pass through the food canal of the mouth parts, escape through the end of the labium when the mosquito feeds on a human being (Fig. 226). On reaching the skin of the human host, they bore through it and enter the blood stream. Thus, the larvae are not introduced into the blood by the mosquito; they bore through the skin by their own power. Upon reaching the lymphatics,

the worms become sexually mature, fecundation occurs, and the females give birth viviparously to enormous numbers of larvae. The new crop of larvae enters the blood stream, thus completing the cycle.

It should be noted that both man and mosquito are necessary for the completion of the life cycle of the parasite. Man is the definitive host; the mosquito is the intermediate host. Since the pathogen is not injected by the feeding punctures of the mosquito but penetrates the skin by its own power, infection is contaminative.

Cattle Ticks and Texas Fever.—Texas fever, or red-water fever, is a widely distributed disease of cattle. It is characterized by a high temperature and a destruction of the red blood corpuscles with the consequent red discoloration of the urine. It is usually fatal although light attacks are not uncommon. It has been one of the most destructive diseases of cattle throughout the southern part of the United States, and the same or a closely related disease is known to occur in other parts of the world.

The disease is caused by a minute protozoan parasite of the red blood corpuscles (*Babesia bigemina*).

Transmission of Texas Fever.—Ticks are responsible for the transmission of Texas fever. No other method of spread in nature is known, although several different species of ticks may serve as vectors [*Boöphilus annulatus* (Fig. 227), *B. dugesii*, *B. discoloratus*, and *Ixodes ricinus*]. When the tick feeds upon infected cattle, it ingests the pathogen along with the blood. The pathogen undergoes a complicated sexual reproduction and multiplies in the body of the tick, large numbers of individuals of a specialized form being thus produced. The new forms of the pathogen enter the ovaries, invade the eggs, and infect the young "seed" ticks before they are born, eventually finding their way into the salivary glands of the new generation. The mature female ticks fall from the infected cattle and deposit their eggs on the ground. The eggs soon hatch, and the young ticks crawl up on blades of grass from which they succeed in getting onto new cattle. When the ticks feed on the cattle, the parasites pass from the salivary glands of the ticks into the blood of the cattle where they infect the red blood corpuscles.

It should be noted that the tick is the definitive host and the cow is the intermediate host. Both the tick and the cow are

necessary for the completion of the life cycle of the parasite. Infection is inoculative, the pathogen being introduced into the blood through the feeding punctures of the vector. The parasite is transmitted congenitally through the egg of the vector, and the disease is not transmitted by the same tick that feeds on the infected cow but by its progeny. Thus, two generations of the vector are necessary for the completion of the transmission cycle.

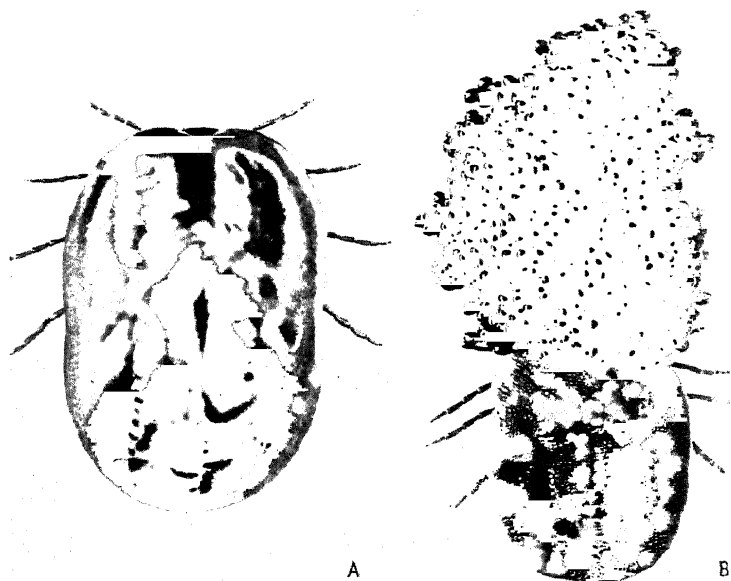


FIG. 227.—The cattle tick (*Boophilus annulatus*), the vector of Texas fever. A, engorged female; B, liberation of eggs. (Photograph reproduced by courtesy of Bur. Animal Indus., U. S. Department Agriculture.)

The Tsetse Fly and the Nagana Disease of Cattle.—The nagana disease is very destructive to cattle in Africa. The hair falls out of affected animals, and the flesh wastes away. There is a running of the nose and mouth accompanied by watery blisters. Blindness eventually develops, and the disease is nearly always fatal. The nagana disease is found only in Africa in regions where the tsetse fly is present. The cause of the nagana disease is a trypanosome (*Trypanosoma brucei*).

Transmission of the Nagana Disease.—This disease is transmitted in nature exclusively by the tsetse fly, *Glossina morsitans* (Fig. 228) although it may be transmitted experimentally by artificially inoculating animals with the blood of an infected animal. The trypanosomes are sucked from the blood stream of affected animals by the fly and, in the body of the insect, undergo a sexual developmental stage requiring a minimum of 10 days. During this 10-day period, the fly is not infective, but after the incubation period is completed the fly remains infective for a long time. The pathogen may be found in the blood of many wild animals that show no marked symptoms of

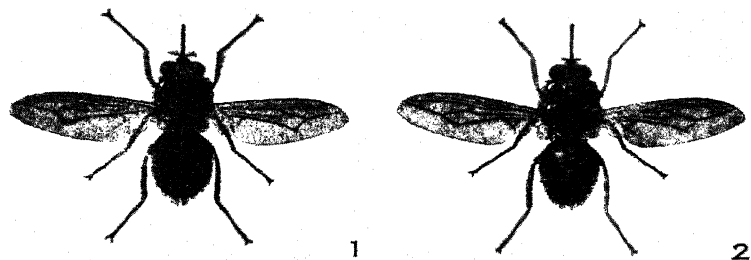


FIG. 228.—Tsetse flies, the vectors of sleeping sickness. 1, *Glossina palpalis*; 2, *G. morsitans*. (From Manson's "Tropical Diseases.")

the disease. These serve as reservoirs from which the pathogen is transmitted to the domesticated cattle.

The insect vector in this case is the definitive host, and the cow is the intermediate host serving as an intermediate host reservoir. Infection is inoculative, the pathogen being introduced directly into the blood stream by the biting fly. The pathogen is not congenitally transmitted in the vector, each insect acquiring it by sucking the blood of an infected animal.

The Tsetse Fly and Sleeping Sickness.—A disease of man very similar to the nagana disease of cattle occurs in many places in Africa. It is indigenous to Africa and has been present for a long time; but, with the development of commerce and travel, it has become more widespread and destructive. The disease is slow and insidious, being practically always fatal. It is characterized by dullness, apathy, and eventually complete lethargy, symptoms that are responsible for the name "sleeping

sickness." The flagellate *Trypanosoma gambiense* is the casual agent (Fig. 229).

Transmission of Sleeping Sickness.—Like the nagana disease, sleeping sickness is transmitted by several species of tsetse fly, principally *Glossina palpalis* (Fig. 228) and *G. tachinoides*. The life history of the parasite, its method of transmission and its host relationships, in all essential respects, are comparable to those of the nagana disease.

Efforts to control the sleeping sickness have emphasized strikingly the importance of a complete knowledge of all aspects of the

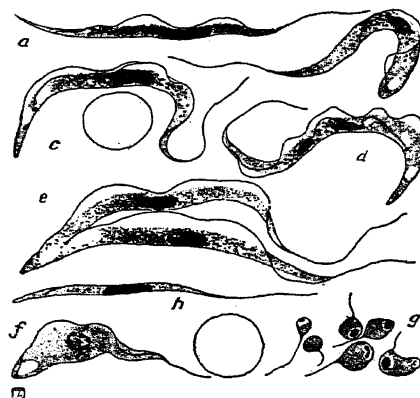


FIG. 229.—*Trypanosoma gambiense*, the cause of African sleeping sickness and transmitted by *Glossina palpalis*. Various forms from the blood and cerebro-spinal fluid. (From Manson's "Tropical Diseases.")

transmission of a disease. With more complete knowledge of the insect vectors, their habits, and geographical distribution, many early conclusions have proved untenable. The most insignificant detail in the life history and biology of the vectors becomes of prime importance in fighting the disease. The marked success that has been attained has resulted from close cooperation between the entomologists and the physicians in their investigations of all aspects of the disease and its insect vectors. A graphic statement of some of the problems involved and how they are being solved has been given by Swynnerton (1936). There are 21 known species of *Glossina* indigenous to Africa, each requiring different vegetative and animal ecology for their opti-

mum development. Remarkable control of the insects and sleeping sickness has been accomplished through ecological modification of the infested areas. The use of properly designed clearings and of plant barriers, especially evergreen barriers, has been very effective. The combined efforts of entomologists, zoologists, botanists, and physicians have been largely responsible for the success attained.

Mosquitoes and Malaria.—Malaria is said to be the worst scourge of mankind. It is one of the most widely distributed

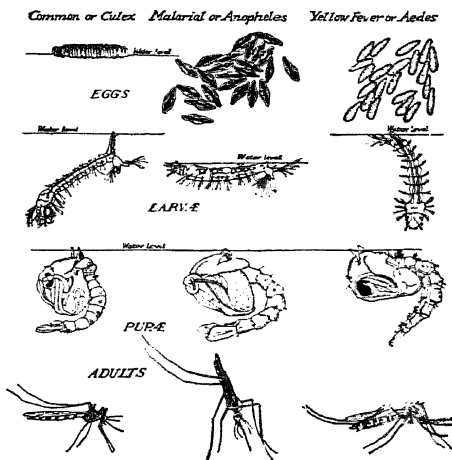


FIG. 230.—The life stages of three types of mosquitoes, *Culex*, *Anopheles*, and *Aedes*. The latter two types are vectors of malaria and yellow fever, respectively. (From "Everyday Problems in Science," Pieper and Beauchamp, copyrighted by Scott Foresman & Company, after Metcalf and Flint.)

diseases affecting man, occurring throughout the tropical and subtropical regions of the world. It is not so fatal as certain other diseases, but its debilitating effect on such a high percentage of the population makes it a serious factor in the health of man. The characteristic symptoms of malaria are intermittent "chills" followed by fever and a chronic languor and debility.

Malaria is caused by minute protozoa that invade and destroy the red blood corpuscles. Three different species are recognized, each causing a distinct type of malaria: (1) *Plasmodium vivax*, causing tertian, or benign, malaria, (2) *P. malariae*, causing quartan malaria, and (3) *P. falciparum*, causing malignant or

estivo-autumnal malaria. The benign, or tertian, malaria is the most prevalent type in North America.

Transmission of Malaria.—Mosquitoes of the genus *Anopheles* (Fig. 230) are the only known agents of transmission of malaria. The common *Culex* mosquito is not a vector. Many different species may transmit the disease, but not all of them transmit it with the same degree of effectiveness. The relation of the mos-

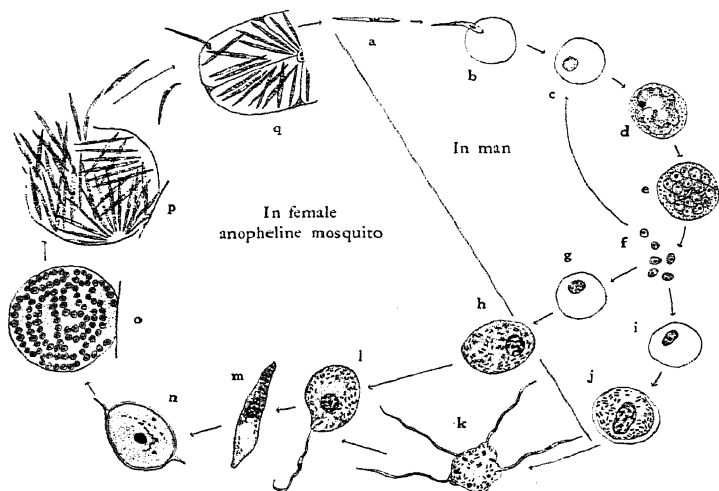


FIG. 231.—The life cycle of the malarial pathogen in man and mosquito illustrated diagrammatically. The sporozoites are introduced into the blood of man from the salivary glands of the mosquito (a). In the human blood stream the parasite multiplies asexually (c to f) forming micro- and macrogametocytes (g to j). When the patient is bitten by a mosquito, these are taken into the body of the insect where the parasite multiplies sexually (o to p); the salivary glands becoming charged with sporozoites (q) completing the cycle. (After Kudo, "Protozoology," second edition, by permission of Charles C. Thomas, publisher.)

quito to malaria has been studied extensively and constitutes what is perhaps the best-known example of insect transmission of a disease. The life history of the pathogen in the human body and in the mosquito has been worked out in great detail and is described adequately in many standard texts. The relationship of the mosquito and *Plasmodium vivax* is, briefly, as follows: In the blood of the human body, the plasmodium inhabits the red corpuscles where it multiplies asexually, causing the cell to

swell and burst, liberating from 15 to 24 *merozoites*, each of which attacks a new red cell and repeats the process in about 48 hours. After several days of asexual multiplication, male and female cells (*micro-* and *macrogametocytes*) are formed. No further change in the life cycle of the pathogen takes place in the human body, the mosquito being essential for further development. When an infected person is bitten by an anopheline mosquito, the sex cells of the pathogen are ingested with the blood (Fig. 231). Fertilization takes place in the stomach of the mosquito, forming a *zygote*. The zygote, or *oökinete*, penetrates the wall of the mosquito's stomach, where it enlarges and transforms into an *oöcyst*. After a time, the enlarged oöcyst bursts and liberates into the body cavity a large number of minute *sporozoites* which invade the salivary glands and are ready to be injected into the blood stream of the next victim that the mosquito bites. The incubation period, or the time required for the completion of the cycle in the insect, is 8 to 14 days. The pathogen may live for several months in the mosquito but does not usually survive the winter in the insect's body, although such survival is possible. The gametocytes may survive for long periods of time in the blood of man, even though they may not cause active symptoms of malaria. Individuals that harbor the gametocytes but have no active symptoms are called "carriers" and are important sources of infection. Birds and other animals are subject to malaria caused by similar but different species of the parasite. They are not intermediate host reservoirs for the malaria that affects man.

Mosquitoes and Yellow Fever.—Yellow fever is among the most virulent diseases of man and has been one of the most dreaded of all epidemic diseases. It is acute and noncontagious, causing prostration, jaundice, and hemorrhages; and the percentage of fatalities is high. The disease was very prevalent for many years in the areas bordering on the Caribbean Sea; and although it was known to occur in other parts of the world, it was generally considered to be of American origin. It was recognized as a disease of civilized regions, apparently being transmitted only by the domestic mosquito [*Aedes aegypti* (L.)], but with the discovery of endemic yellow fever among the natives of Central Africa and South America, in the absence of *A. aegypti*, the whole question was reopened (Carter 1931; Shannon, Whit-

man and Franca 1938). Yellow fever is caused by a filterable virus.

Transmission of Yellow Fever.—Yellow fever, like malaria, is transmitted only by mosquitoes. No other method of transmission is known. For a long time after the first demonstration of mosquito transmission by Reed and his associates in 1900, it was believed that only one species (*Aedes aegypti*) (Fig. 230) was capable of transmitting it. Experimental work on transmission was long handicapped by the absence of any known hosts other than man. In 1928, Stokes, Bauer, and Hudson showed that certain monkeys were susceptible to yellow fever, and with the aid of this new experimental animal Bauer (1928), Shannon, Whitman, and Franca (1938) and others soon demonstrated that mosquitoes other than *A. aegypti* are effective vectors. Now it is known that a dozen or more different species may transmit the virus. Many of these are species inhabiting the jungles, and the virus has been demonstrated in mosquitoes caught in their native jungle habitat.

The incubation period of yellow fever in man is 3 to 7 days. The work of Reed and his associates indicated that in order to transmit the virus the mosquito must feed upon an infected man during the first 3 or 4 days after the initial symptoms of the disease. In monkeys, however, the virus persists in the blood stream throughout the course of the disease and may be acquired by the mosquitoes at any time. An incubation period of 9 to 14 days in the mosquito is required before the virus can be transmitted. The nature of this incubation period and what changes, if any, occur in the virus are not known. After the incubation period, the mosquito is capable of transmitting the virus as long as it lives. A single bite of one infected mosquito is sufficient to produce infection. Although congenital transmission of the virus in the mosquito has been claimed by some workers, the more recent evidence has failed to support the claim.

As soon as it was learned that yellow fever was transmitted by mosquitoes, efforts were made to control the disease by exterminating the mosquitoes. So successful were these efforts that the disease practically has been eliminated from the more civilized regions of America. Equally successful results were obtained in the Philippine Islands, but only after a careful study was made of the habits of the mosquitoes responsible for its spread. The

active vector in the Philippines has breeding habits so different from those of *A. aegypti* that the measures used in Cuba and Panama would not have been effective. In fact, the breeding habits of the different species vary widely, and the control of yellow fever in any region must be based largely on a complete knowledge of the life history and breeding habits of all the mosquitoes capable of transmitting the virus.

Mosquitoes and Dengue.—Dengue, or breakbone fever, is prevalent throughout many tropical and subtropical regions of the world. It is characterized by headache and severe body and limb pains but is rarely ever fatal. It is infectious and often occurs in epidemic proportions. There was a severe epidemic in Texas and neighboring states in 1922. The cause of the disease is not known definitely, but the infectious agent has many of the characteristics of a virus.

Transmission of Dengue.—Dengue is transmitted by mosquitoes, the most important species being *Aedes aegypti* and *A. albopictus*. The method of transmission is similar to that of yellow fever in all essential respects. The virus appears to be in the blood stream and can be acquired by the mosquitoes only during the first 3 days of the attack. An incubation period of 8 to 11 days in the mosquito is necessary before transmission is possible. The mosquito retains the virus for the length of its life, but there is no evidence of congenital transmission by the mosquito. Monkeys are susceptible but show no clinical symptoms, although they may serve as animal reservoirs.

Fleas and Bubonic Plague.—Bubonic plague is an acute infectious disease affecting both man and rodents. It is a bacterial disease caused by *Pasteurella pestis* (Lehman and Neuman) Bergey *et al.* In man, it is characterized by swellings of the lymphatic glands, usually on the neck, armpits, or groins, often accompanied by black spots or blotches under the skin. There is sometimes a secondary congestion of the lungs causing the so-called "pneumonic" form of the disease. Plague is highly fatal to both man and rodents. It is the Black Death of the Middle Ages and was the cause of the Great Plague of London in the seventeenth century. The devastating nature of its epidemics made it one of the most dreaded of the diseases of man.

Transmission of the Plague.—Plague is primarily a disease of rodents; rats, mice, ground squirrels, and many other species are

affected. It may be transmitted by direct inoculation, but in nature the principal agents of transmission are fleas. Any flea that feeds upon both rodents and man may transmit the disease to man; and most of the fleas that feed upon rodents will, under certain conditions, feed on man. Man is most frequently affected when rats die of plague and the fleas leave the dead rats and infest man. Because of the life habits of rats which infest human habitations, they are the most dangerous of the rodents in spreading the disease among man. The black rat (*Rattus rattus*) and the brown rat (*Rattus norvegicus*) are species most concerned. More than a dozen species of fleas may transmit plague, but the Indian rat flea (*Xenopsylla cheopsis*) is the most universally important one.

The flea is not a true intermediate host for the plague bacteria, for no cyclic development occurs within its body. Therefore, it is not the only possible agent of transmission. Artificial direct inoculation is effective, and the disease can be transmitted by any agency that is capable of introducing the bacteria into the blood. However, in nature the rat flea is the most effective vector and the only one of any great significance.

The bacteria multiply in the bodies of the fleas that have fed on infected rats. The bacteria may pass through the intestinal tract in a viable condition, contaminate the skin, and gain entrance through wounds made by scratching. The bacteria also may be inoculated directly under the skin in the feeding process by certain individual fleas in which the stomach opening is clogged so that blood and bacteria are regurgitated and forced back into the feeding wounds. Transmission, therefore, may be either contaminative or inoculative.

The control of plague is based upon the destruction of the rat and its fleas. The disease is commonly spread to new regions by rats carried on ships, the infected rats leaving the ship and starting an epidemic on shore. Ships from infected ports are fumigated not to kill the germs directly but to kill the rats and their fleas. Control by this means has been very successful in the more civilized countries, but in some of the less advanced tropical countries the disease still persists. Also, where the native wild rodents have become infected they serve as potential reservoirs from which the disease may spread to the semidomesticated rats and from them to man.

Lice and Typhus Fever.—Typhus fever is a disease of ancient origin and of wide distribution. It is most prevalent in temperate and cold climates and is closely associated with crowded and unsanitary living conditions. It is usually prevalent among armies in wartime. The mortality varies from 5 to 50 per cent. Typhus fever is caused by *Rickettsia prowazekii*, a minute intracellular microorganism found in the epithelium of the intestines of lice as well as in the blood of man when affected with the disease.

Transmission of Typhus Fever.—Lice (*Pediculus humanus*) are the agents of typhus transmission, the disease having been transmitted experimentally by lice from monkey to monkey. No other insect vector is known. There is a required incubation period of 8 to 9 days in the louse before it can transmit the disease. Transmission may be inoculative through the feeding punctures of infected lice, or contaminative by infected excreta of infected lice, the pathogen entering through abrasions in the skin such as those made when the infested person scratches himself. The crushed bodies of infected lice also may serve as a source of inoculum. There are some indications that the pathogen may be congenitally transmitted in the louse, but the evidence is not conclusive.

Lice and Trench Fever.—Trench fever is a disease similar in many respects to typhus fever, but much less fatal. It was prevalent among the soldiers of the World War and was first recognized as a distinct disease at that time. It is caused by *Rickettsia quintana*, a microorganism found consistently in the stomach of infected lice.

Transmission of Trench Fever.—Trench fever is infectious and may be transmitted by artificial inoculation but in nature is transmitted almost exclusively by lice (*Pediculus humanus*). Transmission may be inoculative, the pathogen being introduced into the blood through the feeding of infected lice, or contaminative through the excreta of lice coming in contact with abrasions in the skin. An incubation period of 5 to 9 days in the lice is necessary before transmission is possible by either means. The pathogen is not congenitally transmitted through the eggs of the lice.

Flies and Typhoid Fever.—Typhoid fever is an enteric infection caused by *Eberthella typhosa* (Zopf.) Weldin. The pathogen is

primarily a parasite and does not survive very long outside of the human body, but it may survive long enough to be transmitted through food and drinking water. It is found principally in the intestines, urinary bladder, gall bladder, and blood stream. The bacteria are voided in great numbers in the excrement and urine of affected individuals. Infection follows ingestion of the bacteria, which occurs most frequently as a result of using contaminated food or drinking water.

Transmission of Typhoid Fever.—Because of the method of infection and the prevalence of the pathogen in the excretory products of typhoid patients, the transmission of typhoid fever is accomplished largely by the contamination of food and drink with bacteria from exposed excrement or untreated sewage. This contamination may be brought about in several ways. One of the most common is through direct contamination of the water supply from fresh excrement or sewage; another common method is by direct contamination of food or milk through handling by a chronic carrier, a person who has recovered from typhoid fever but continues to pass the pathogenic bacteria in the feces or urine. Fortunately only a small percentage of recovered patients are carriers. The common housefly (*Musca domestica* L.) also has been incriminated as a vector of the typhoid bacillus. Houseflies are common inhabitants of sewage and exposed human feces; they not only walk over the contaminated material but feed upon it and ingest the bacteria; they also breed in contaminated material and are common visitors to exposed food and milk. It has been demonstrated that the typhoid bacteria may survive within the puparia of the housefly (Bacot 1911) but in view of the abundant opportunity for contamination in other ways it is not known how important this fact is in the epidemiology of the disease. Its chief significance would probably be in preserving the bacteria over an unfavorable period.

The housefly, in addition to being a vector of typhoid fever, has been incriminated along with closely related species as a vector of a number of other animal diseases. In all cases, the method of transmission is essentially the same, infection being contaminative and resulting from the pathogen borne either externally or internally by the vector. In the latter case, the contamination follows regurgitation or defecation.

A BRIEF COMPARISON OF INSECT TRANSMISSION OF PLANT AND ANIMAL DISEASES

The foregoing examples are fairly representative of the most important insect-transmitted diseases of animals and offer a basis for comparison with insect-transmitted plant diseases. To facilitate the comparison, a tabular summary of the phenomena of insect transmission, as represented by these diseases, is given in the appendix, along with similar tables for representative insect-transmitted plant diseases. It will be noted that there are many striking similarities in the phenomena associated with insect transmission of the two kinds of disease. There are also a number of significant differences. In general, the same kinds of pathogen are involved in both groups of insect-transmitted diseases. The virus diseases constitute the largest group of insect-transmitted plant diseases, followed by the fungus and bacterial diseases, with only a few caused by protozoa. Diseases caused by protozoa lead the list of insect-transmitted animal diseases, followed closely by *Rickettsia* and viruses and a smaller number of bacterial diseases. There appear to be no fungus diseases of animals that are transmitted to any extent by insects.

There is a wide variation in the specificity of the relationships between vector and disease in both groups. In plants, the more highly specialized relationships are found in the virus diseases which are, for the most part, transmitted by the sucking insects. Similar specialized relationships are found in the virus diseases of animals and in many animal diseases caused by protozoa that are transmitted only by certain bloodsucking insects. The highest degree of specialization is found in the latter group where the insect vector is often a definitive host and is necessary for the completion of the life cycle of the pathogen. Although there are several plant diseases in which the insect vector is obligatory, the definitive-host relationship is not known, with the possible exception of one or two incompletely studied cases among the endophytic protozoa. As an example may be mentioned *Phytomonas davidi* causing a flagellosis of *Euphorbia*. This phytopathogenic flagellate is transmitted by *Stenocephalus agilis* which Franca (1920a) claims is a definitive host, having seen what he interpreted as a sexual stage of the flagellate in the insect.

In so far as is known, animal diseases, such as typhus fever and trench fever, are transmitted in nature by a single species of insect. Among plant diseases, the curly top of sugar beet, peach yellows, cranberry false blossom, and a few other virus diseases are each transmitted by a single species of insect.

Malaria is transmitted only by mosquitoes of the genus *Anopheles* whereas yellow fever is transmitted by several genera of mosquitoes. Similar examples of group specificity are found among the plant diseases as represented by corn streak, transmitted by three species of *Cicadulina*, and by potato leaf roll, transmitted by several genera of aphids.

The necessity for an incubation period in the body of the insect vector is a phenomenon common to transmission of both plant and animal diseases. In the case of protozoal pathogens, where a cyclic development is known, the nature of the incubation period is obvious, but the nature of the incubation period in vectors of virus diseases of both plants and animals is not definitely known.

Congenital transmission of the pathogen occurs in vectors of both plant and animal diseases but is not a common phenomenon in either group. The protozoal pathogen of Texas fever is congenitally transmitted by the cattle tick, and the virus of the dwarf disease of rice is transmitted in a similar manner to successive generations of the leaf hopper (*Nephotellix apicalis*).

In reference to both plant and animal diseases, a knowledge of the phenomena of insect transmission has aided greatly in the control of the diseases concerned. The limits of possible control, however, have not been reached. There is much yet to be gained through more extensive studies of insect transmission of plant diseases, and those who investigate the field will profit greatly by acquainting themselves with what has been accomplished in the study of insect transmission of animal diseases.

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CHAPTER XVII

METHODS OF INVESTIGATING THE RELATION OF INSECTS TO THE SPREAD AND DEVELOPMENT OF PLANT DISEASES

Research in the field of insect transmission of plant diseases does not differ from that in any other field of biology in so far as the basic principles are concerned. The same general procedures are necessary. Observation, experimentation, analysis and interpretation of data, constructive imagination for the building of working hypotheses, and inference, both deductive and inductive, are all essential. The technical methods involved, however, are unique in that they embrace those of two distinctly different fields of research, namely, phytopathology and entomology. Workers in each of these two fields have developed a series of technical methods particularly adapted to their needs, and each group of workers is relatively unfamiliar with the techniques of the other group. Few workers trained primarily as plant pathologists have had much experience in handling and rearing insects. The average entomologist likewise has had little experience with the bacteriological and mycological techniques that are basic necessities in the study of plant diseases. The lack of familiarity with either type of technique is a great hindrance in the study of insect-transmission problems and often discourages an investigator from doing work in the field.

The investigation of problems in insect transmission of plant diseases is often done cooperatively by one or more workers from each field. This is desirable because of the added experience and knowledge of techniques brought to bear on the problem. Theoretically, the problem of techniques could be solved by cooperation in which the work requiring entomological technique would be done by the entomologist and that requiring phytopathological technique by the phytopathologist. In practice, however, such strict division of labor is rarely successful. Cooperative research organized on this basis often is little more

than regimentation, and when research is regimented the spirit of adventure is lost. Experience has shown that regimented research is rarely, if ever, productive research. It is sometimes stated that among social insects, such as bees, ants, and termites, the welfare and personal interests of the individual are completely subordinated to the welfare of the group and that a similar social organization should be striven for in human endeavors. This stage of social development, however, has not been reached in human society, and complete subordination of personal interest is not found often in cooperative research. Because of the personal equation involved, a less highly socialized and more flexible plan of cooperation is more likely to be successful. Moreover, it is frequently impossible to make sharp divisions of the work on the basis of technique. More often than not, the two kinds of technique and the problems being investigated are so closely interrelated that no practical division is possible.

It is beyond the scope of this book to present an extensive discussion of the techniques of plant pathological and entomological research. In this chapter, certain general principles peculiar to the problem of insect transmission will be discussed briefly, and a few items of technique that are directly applicable to the subject will be described. The principles and techniques of research in plant pathology have been treated by Riker and Riker (1936) and Rawlins (1933); and Peterson (1934, 1937) has published well-illustrated manuals of entomological equipment and methods. These publications will be found very helpful by the student of insect transmission of plant diseases.

KOCH'S POSTULATES

Before a satisfactory study of insect transmission of a plant disease can be made, it is necessary to learn as much as possible about the nature and cause of the disease that is transmitted. If the disease is caused by a microorganism, it is highly desirable that the nature and identity of the organism be known and its pathogenicity proved beyond doubt. In the last quarter of the nineteenth century, shortly after the discovery that bacteria could cause animal diseases, many species of bacteria were reported as causing disease but adequate proof of pathogenicity was often lacking. In an effort to discourage this practice, Koch (1883) published a statement of certain requirements

that he thought should be met before a claim of pathogenicity is made. These requirements have been accepted in principle by both animal and plant pathologists. They are generally known as "Koch's postulates." Every investigator who works with insect transmission of a disease should be familiar with them. They may be stated briefly as follows:

1. It must be demonstrated that the microorganism occurs consistently associated with the disease in question.

2. The microorganism must be isolated in pure culture and adequately described.

3. The disease, with characteristic symptoms, must be reproduced by artificial inoculation of a suitable suspect with the microorganism in pure culture.

4. The microorganism must be reisolated from the inoculated suspect and identified with the one used for inoculation.

Obviously, it has not been possible to apply these postulates to those diseases caused by obligate parasites and by virus diseases, for no technique for growing these pathogens in artificial culture is known. Other criteria of proof must be used for such diseases. The relation of Koch's postulates to virus diseases, with particular reference to animal diseases, has been discussed by Rivers (1937), who points out that the spirit of the postulates can be met even though the virus cannot be cultivated in artificial media.

Complete proof, in the spirit of Koch's postulates, that a virus is the cause of a plant disease would require the demonstration of the following facts:

1. That inoculum taken from the diseased plant and freed from ordinary microbes by filtration or other means will cause the disease when inoculated into healthy plants.

2. That the infectious agent was neither fortuitously present in the inoculated plant nor introduced from some extraneous source (this can be demonstrated by adequate checks in the inoculation experiments).

If the disease is transmitted by insects and not by artificial sap inoculation, complete compliance will require transmission by feeding the insect vector on filtered extracts from the diseased plants and subsequently letting it feed upon healthy plants. Methods useful for this have been described by Carter (1927, 1928), Severin and Swezy (1928), Storey (1932), Bennett (1935),

Bennett and Esau (1936), and others. Several different types of insect cage adapted to feeding the insects on filtered plant extracts have been used. All make use of a thin membrane which separates the liquid from the insects and through which the insects feed. The feeding cage used by Storey (1932) in feeding the leaf hopper (*Cicadulina mbila*) on filtered extract from corn affected with streak is shown in section in Fig. 232. The membrane used is that known as "Baudruche capping skin" and has proved to be very satisfactory for this purpose. The filtered juices are placed in the upper section and the leaf hoppers in the lower. The cage is set up so that the strongest source of light for the hoppers is seen through the membrane. By virtue of the light, the insects are attracted to the membrane, through which they feed upon the drops of filtered extract. The cages used by Bennett (1935) for feeding the sugar-beet leaf hopper on extracts from beets affected with curly top are of similar construction, but the insects were placed in the upper chamber and drops of the liquid were placed on the underside of the membrane. Other modifications of this feeding cage have been described by Hamilton (1930). Fife (1932) has devised a method in which paraffin sections cut 60 microns thick are substituted for the usual membrane. When small drops of liquid are placed on the paraffin, the insects (*Eutettix tenellus*) readily penetrated the thin slice of paraffin and fed upon the liquid.

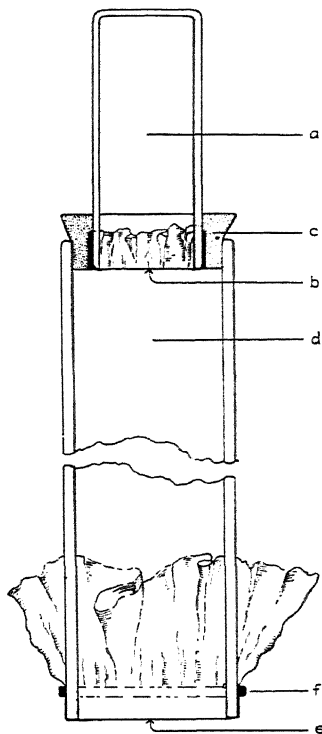


FIG. 232.—A convenient feeding cage for feeding-sucking insects on filtered plant extracts or other solutions. *a*, Glass tube containing the feeding fluid; *b*, membrane of Baudruche capping membrane; *c*, rubber stopper; *d*, glass tube 8 inches long and 1 inch in diameter in which the insects are caged. *e*, muslin cover; *f*, rubber band. (After Storey.)

By virtue of the small drops of liquid used, this method was valuable in determining the pH of the salivary secretions of the insect and optimum pH of artificial nutrient solutions.

The liquid used by Storey for feeding the leaf hoppers was prepared by crushing diseased corn plants and squeezing out the juice, filtering it first through paper pulp, and mixing the filtrate with an equal part of a 20 per cent sucrose solution. This fluid was then mixed with a suspension of test bacteria and filtered through Chamberlain and Berkfeld filters. The filtrates were tested for sterility and then used for feeding the leaf hoppers, which, after feeding on the liquid for 24 hours, acquired the virus and transmitted the disease to healthy plants by feeding. This method is not only valuable for proving the filterability of viruses that are transmitted solely by insects but is useful also in the study of other aspects of the problem. The method has been used by Bennett (1935) and Bennett and Esau (1936) in studies of the properties of the curly-top virus and the movement of the virus in the plant. In these studies, an improved technique of preparing extracts for use in feeding the leaf hopper was devised.

Juice was expressed from the tissues to be tested and added to an equal volume of 95-per cent alcohol. The resulting precipitate was thrown down by centrifugation, washed once with 50-per cent alcohol, dried, mixed with a volume of 5-per cent sugar solution equal to the original volume of juice, and centrifuged, and the supernatant liquid was used to feed non-viruliferous leaf hoppers.

The more recent work of Vinson and Petre (1929, 1931), Stanley (1936*a*, 1936*b*), Thornberry (1938), Vinson, McReynolds, and Gingrich (1939), and others, in which techniques have been devised for isolating virus proteins in crystalline form, may provide a more direct way of meeting the requirements for proof that diseases are caused by viruses. The techniques for isolating the virus proteins have not yet been standardized adequately, and continued study will doubtless result in many refinements over the ones in use at the present time. The original method used by Stanley (1936*a*), who first isolated the virus protein in crystalline form, was soon modified and improved so that the yield of crystalline protein was practically doubled (Stanley 1936*b*). The original method consisted in the precipitation with ammonium sulphate of the globulin fraction from a dilute

disodium phosphate extract of frozen, macerated, infected plants, and the removal of the coloring material by treatment with lead subacetate and celite. In the improved method, lead subacetate is not used, the precipitation and clearing being accomplished with ammonium sulphate, celite, and calcium oxide. The essential steps in the method are as follows (Stanley 1936b):

Tobacco plants affected with mosaic are cut, frozen, and put through a meat grinder, and the pulp is extracted twice in a fruit press with 0.1 m. sodium phosphate at pH 7. The two extracts, each of which should equal about 70 per cent of the volume of the original pulp, are combined, and ammonium sulphate is added at the rate of 300 grams to 1,000 cubic centimeters of extract. The precipitated globulin is collected by gravity filtration through paper and when analyzed should yield almost 2 grams of protein to each 1,000 cubic centimeters of original extract. The precipitate is dissolved in 0.1 m. sodium phosphate solution at pH 7 and filtered, with suction, through a thin (0.5 centimeter) layer of celite (Hyflo Standard-cel) on a Buchner funnel. The celite filter cake is washed with 0.1 m. sodium phosphate at pH 7, and the protein in the filtrate is precipitated with ammonium sulphate, added at the rate of 20 grams to 100 cubic centimeters of filtrate. The precipitated protein is collected on filter paper and dissolved in 0.1 m. sodium phosphate at pH 7. About 8 to 11 per cent by weight of ammonium sulphate is added, producing a slightly turbid solution which is filtered through a thin layer of celite, with suction, on a Buchner funnel. The celite filter cake is washed with 8 per cent ammonium sulphate solution and this filtrate added to the previous filtrate. Enough ammonium sulphate is added to the combined filtrates to bring the ammonium sulphate concentration up to 20 per cent by weight. The precipitated protein is then removed by filtration through a thin layer of celite on a Buchner funnel. All the virus protein is retained on the celite and can be removed by extracting three times with 0.1 m. sodium phosphate solution at pH 7. The filtrate is adjusted to pH 4.5 by the addition of 3 m. sulphuric acid and filtered through a thin layer of celite. The celite filter cake is then suspended in 300 cubic centimeters of water, the reaction being adjusted to pH 8 by the addition of an aqueous suspension of 5 per cent calcium oxide, and filtered with

suction through celite on a Buchner funnel. The celite filter cake is extracted with water at pH 8 and should yield a practically colorless opalescent solution. The protein is then crystallized by adding to 750 cubic centimeters of the solution 75 grams of solid ammonium sulphate, then 6 cubic centimeters of a solution of 5 per cent glacial acetic acid in 0.5 saturated ammonium sulphate, and finally 20 cubic centimeters of saturated ammonium sulphate solution.

The procedure above may vary with the plants used. In the extraction of the tobacco-mosaic virus, plants inoculated when they are young and extracted about 4 weeks after inoculation give higher yields of pure crystals than plants inoculated when old. The pH of the solutions during extraction should be carefully controlled and should never be allowed to become greater than pH 8.5. In the more alkaline solutions the proteins are denatured with loss of infective power.

Thornberry (1938) has described a modification of Stanley's method in which the previously frozen pulp is heated at 40°C. before extraction.

Vinson, McReynolds, and Gingrich (1939) recommend the use of solid sodium sulphate instead of ammonium sulphate for salting out the virus in crystalline form. They point out that the use of a nitrogenous salt to salt out a nitrogenous body is not desirable because of the danger of including some of the salting agent in the precipitate. They believe that the higher nitrogen content (20 per cent) reported for earlier isolations of the virus as compared with later estimates (16.7 per cent) can be explained on the basis of admixtures of ammonium sulphate which has a nitrogen content of 21.2 per cent.

Solid sodium sulphate may be used for salting out the virus from fractions purified by any one of several methods. The initial precipitation from the expressed juices may be made with lead acetate (Vinson and Petre 1931), with saffranin (Vinson 1932), with acid (Vinson 1936), or with solid anhydrous sodium sulphate following acidification to pH 4. The precipitate is then dispersed in a dilute solution of sodium phosphate at pH 7. After adjusting to pH 4, the virus is precipitated by adding the solid sodium sulphate.

Most of the ash may be removed from the precipitate by washing with dilute acetic acid at pH 3.5, and the brown pigment is

readily removed by shaking a neutral phosphate dispersion of the crystalline virus with a volume of ether equal to about 30 per cent of the suspension and removing the ether by centrifuging.

The chemical and physical properties of the viruses isolated by any of these methods may be studied, and the purified virus may be used in direct inoculation studies. So far, the only viruses that have been obtained in crystalline form are readily transmitted by artificial sap inoculation. Viruses that have been transmitted only by insects or by grafting may offer greater difficulties.

The proof of the virus nature of those diseases which have been transmitted only by grafting obviously does not meet the requirements stated above. These diseases are assumed to be caused by viruses merely on the basis of general similarity to other known virus diseases. No satisfactory method of complying with Koch's postulates has been devised for this group of diseases. If no insect vectors are ever found for these diseases, the isolation of the virus proteins and their use for reinoculation possibly may provide a means of proving their true nature.

RULES OF PROOF FOR INSECT TRANSMISSION

Just as bacteria have been described without adequate proof, as being pathogenic, so have many species of insect been reported, with insufficient evidence, to be vectors of plant diseases. Incomplete reports often remain unverified and lead to confusion in the literature. It is desirable, therefore, that certain definite requirements be met before it is concluded that an insect is a vector of a plant pathogen. It is believed that the following requirements constitute the minimum for adequate proof of insect transmission of a plant disease.

1. A close, although not necessarily a constant, association of the insect with diseased plants must be demonstrated.
2. It must be demonstrated that the insect also regularly visits healthy plants under conditions suitable for the transmission of the disease.
3. The presence of the pathogen or virus in or on the insect in nature or following visitation to a diseased plant must be demonstrated.
4. The disease must be produced experimentally by insect visitation under controlled conditions with adequate checks.

When evidence is presented that an insect is a vector of a plant disease, it should always meet the above four requirements.

It is equally necessary that the postulates of Koch be observed, for if there is an error in the diagnosis of the disease the remainder of the evidence is invalidated.

SUPPLEMENTARY DATA

When an insect is incriminated as a vector of a plant disease, its relative importance in relation to other agents of transmission always should be determined in so far as possible, for only when this is known can the discovery be utilized effectively in a practical plan for controlling the disease. It is important also to determine whether the insect is a vector of primary inoculum or of secondary inoculum or of both. An insect that is a vector of primary inoculum may be of much greater significance in the epiphytology of a disease than one which is concerned only with the dissemination of secondary inoculum.

In Chap. III, it was shown that insects may influence the development of plant diseases in several ways. The insect may serve only as an agent of dissemination of the pathogen; or it may also be responsible for inoculation, for ingression, and for preservation of the pathogen over periods of unfavorable environment. The extent to which the insect performs each of these various functions should be determined.

The ability of a suspected vector to transmit a disease should be tested in all stages of development. It has been shown that the ability of certain vectors to acquire the inoculum, or to transmit it, varies greatly with age. For example, *Thrips tabaci*, the vector of spotted wilt, cannot acquire the virus by feeding as an adult on infected plants but acquires it readily in the larval stage. Having acquired the virus as a larva, the insect remains infective through metamorphosis and transmits the virus as an imago. As another example may be mentioned the tingid bug (*Piesma quadrata*), the vector of sugar-beet leaf curl, which is unable to transmit the virus in the larval stage but readily acquires and transmits it as an adult. The reverse of this situation exists in the case of certain toxicogenic insects which are toxiniferous only in the nymphal stage. Thus it has been demonstrated that psyllid yellows of potatoes is caused only by the nymph of *Paratrioza cockerelli*, the adult psyllid being unable to cause the disease.

It has been demonstrated by several workers that there are strains within a species of insect, some of which transmit a virus readily whereas others are totally unable to do so. The recognition of this fact makes it imperative that more than one strain of a species be tested, especially when negative or inconsistent results are obtained with a suspected vector.

TAXONOMIC PROBLEMS

The importance of correct identification of the insects studied as possible vectors cannot be overemphasized. Much confusion has been caused by incomplete or incorrect identification of newly discovered vectors. Insect taxonomy is a matter for the specialist, and critical identification of species on which significant data are published should not be attempted by the novice. The best plan is to submit carefully chosen specimens to a well-recognized authority on the particular group of insects concerned. When the data are published, the authority responsible for the identification should be given. Smith (1937) has discussed the importance of this aspect of the problem and has emphasized the need for permanent reference collections of insect vectors of plant diseases. He states:

To be certain of the specific identity of an insect vector is just as important in the problems of identification and means of dispersion of a given virus disease as in those problems presented by any other injurious insect. Whenever, in pathological experiments, the ability of an individual insect to transmit a particular virus has been demonstrated, the insect involved seems to possess a value to the virus problem approaching that of a type as designated by a taxonomist describing a new species, and representatives of such specimens should be preserved for future reference. This is especially important when there is any doubt as to the identity of the species represented, or when it belongs to a group in which related species might be confused.

In the publication of the results of transmission tests an author could indicate the location of representatives of the experimental insects together with the accession number or other data by which the material may be located. The insects selected for preservation could be taken from any of the controlled experiments where positive results were obtained, particularly if from pure-line-bred stock. Whenever insects are submitted to the taxonomist for identification, ample material should be included, so that he will have no excuse for not returning

duplicates if the experimenter is interested in having them for reference or exchange.

THE IMPORTANCE OF FIELD OBSERVATIONS

The methods used in investigating problems of insect transmission vary with the diseases and insects being studied. Some problems are well adapted to study in greenhouse and laboratory, while others must be investigated under field conditions. Whenever possible, greenhouse and laboratory studies should be supplemented by field experiments under conditions as nearly normal as can be obtained. In the investigation of any problem of insect transmission, extensive and thorough observations of the disease as it develops in nature and of the habits of the associated insects are of paramount importance. Casual observations at irregular intervals are not sufficient to give a complete picture of the disease and the phenomena associated with its transmission. The best results are obtained when one, so to speak, lives with the problem until he knows all its ramifications. Observations apparently insignificant at the time often prove to be keys to the solution of a difficult problem. This is true of almost any biological problem but applies particularly to investigations of this kind in which the worker is likely to be relatively unfamiliar with the behavior of either the disease or the associated insects.

GREENHOUSES AND CAGES

Since virus diseases of plants are so universally transmitted by insects, the phenomena of insect transmission has assumed a very prominent place in their study. This has resulted in the development of a number of special techniques for the purpose. Most virus diseases are well adapted to greenhouse study, and much of the work on insect transmission is done in greenhouses.

The two most important principles to be observed, in the study of insect transmission of virus diseases in greenhouses, are that an adequate supply of vigorous normally growing plants be available and that means be provided for adequate insect control. Normal vigorous plants are necessary because of the extreme importance of symptoms in the study of virus diseases. Unless the plants are vigorous and growing normally, symptoms lose much of their value as criteria in virus studies.

The necessity for insect control is obvious but is sometimes disregarded. Reliable results cannot be obtained in greenhouses overrun with aphids, white flies, red spiders, thrips, or other arthropods. A regular routine of fumigation is necessary. In order to preserve cultures of insects for study, more than one house or compartment must be available. The standard nicotine fumigation for aphids and hydrocyanic acid gas for other insects are the usual methods.

The plan of construction most commonly used for greenhouses is not well adapted to the study of insect transmission. A greenhouse with several insect-proof compartments opening upon a central corridor is one of the most satisfactory arrangements.

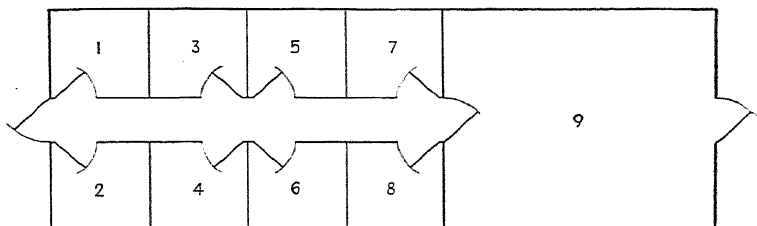


FIG. 233.—Plan of a greenhouse of a type desirable for work with insect transmission of plant diseases. The separate compartments allow separation of diseased and healthy plants and different kinds of insect. It also allows for partial fumigation and other necessary operations. (After Samuel, Bald, and Pittman.)

The plan of such a greenhouse designed for this kind of work is shown in Fig. 233. All ventilators should be provided with screens to prevent the entry of insects from the outside and the escape of experimental insects.

In studying insect transmission of a disease, it is obviously necessary to control all other possible means of transmission. For this reason, it may be necessary to use sterilized soil. Many different kinds of plant pathogen may survive in the soil and serve as unexpected sources of infection. Moreover, many insects that may serve as possible vectors may be found in non-sterilized soil.

Some of the viruses are highly infectious, but most of them require wounds for infection. The necessary wound in some cases may be the mere breaking of a trichome. Many fungi and bacteria also require wounds for infection. Therefore, it usually is important that experimental plants be handled with

extreme care to avoid accidental infection as a result of unnecessary wounding.

The different types of cage used in rearing insects are so numerous that no attempt is made to describe them here. One beginning work in this field will do well to consult the manuals published by Peterson (1934, 1937). Wire screen, cheesecloth, and glass are the most common materials used for making cages. A cloth, originally used for shading tobacco and now used extensively in commercial aster growing, is good for this purpose. Cellophane is proving a very satisfactory material in the construction of cages that are not too much exposed to the weather.

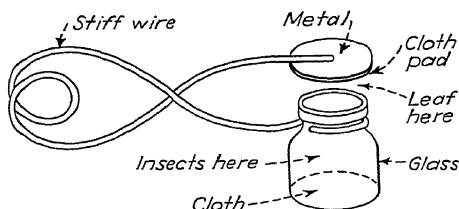


FIG. 234.—A simple type of microcage designed for feeding a small number of insects on a localized area of a plant. The cage is illustrated in the sprung or open position. (After Peterson.)

The variety of useful cages that can be made from this material supplemented with "Scotch tape," gauze, and other easily obtainable materials is limited only by the ingenuity and dexterity of the worker. Cellophane has the advantage of being relatively cheap, light, and very transparent. When plants are grown under cages, the reduced light intensity is often a limiting factor in normal growth. Peterson (1934) has compared the light transmission of various materials used in making insect cages. Cellophane had the greatest light-transmitting power of all the materials tested. When greater strength is desired, screen wire infiltrated with cellophane may be used, but the light transmission is reduced by nearly one-half.

When it is desirable to feed insects upon localized regions of a plant, some kind of microcage is necessary. The form of the cage will be determined by the insects and plants to be used. Convenient microcages have been described by F. F. Smith (1931), K. M. Smith (1932), Samuel, Bald, and Pittman (1930), Giddings (1939), and others. A satisfactory type of microcage

is illustrated in Fig. 234. The cage designed and used by Giddings (1939) for feeding leaf hoppers on individual sugar beet leaves is shown in Fig. 235.

For the collection of small insects and for transferring them from cage to cage, a suction collector of the type illustrated in Fig. 236 is indispensable. Several modifications of this device have been described, but the type illustrated appears to be the most satisfactory for general use.

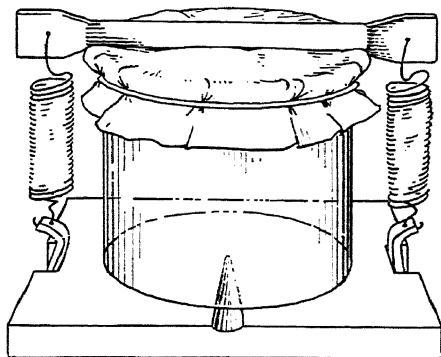


FIG. 235.—A microcage designed and used for feeding a small number of leaf hoppers on individual sugar-beet leaves. The cage is composed of a small glass cylinder closed with gauze at one end and held firmly against a small piece of glass by two springs and specially made metal clamps. The edges of the glass are ground to prevent bruising or cutting, and the glass plate is grooved to take the leaf mid-rib or petiole. (After Giddings.)

INSECT TRAPS

In the study of insects in relation to the development of diseases, it is often desirable to obtain data on their abundance and movement under normal field conditions. Some insects winter in definite restricted localities and spread gradually into new areas as the growing season advances, whereas others appear suddenly as a result of migration. Still others are dispersed by wind currents. The abundance of a given insect vector at a given time and its method of dispersal are obviously significant in the epiphytology of the disease. Insect traps of various kinds are used for obtaining such data.

One of the most useful types of insect trap for determining the prevalence of insects is the light trap. In this trap, the insects

are caught at night, being attracted by a light and entrapped in a cyanide jar. Many different designs of varying degrees of

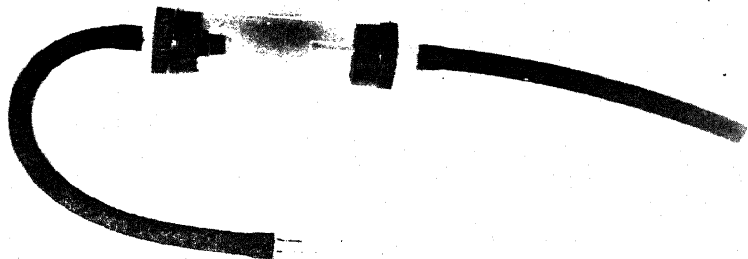


FIG. 236.—A simple and practical insect collector of the suction type that is indispensable for handling individual insects of the kind usually serving as vectors of plant diseases. Its structure and operation are self-evident from the picture.

complexity have been used. A simple, but satisfactory light trap for general use is illustrated in Fig. 237. Other designs to

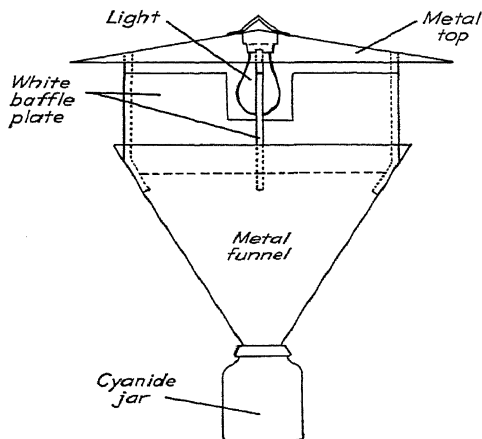


FIG. 237.—A diagram showing the structure of a simple but satisfactory light trap. (After Peterson.)

meet special demands are described by Peterson (1934). The cyanide jar is prepared by placing potassium or sodium cyanide,

about $\frac{1}{4}$ inch deep, in the bottom of the jar and then adding a thin layer of moist plaster of Paris, which soon hardens. Enough hydrocyanic acid gas will diffuse through the plaster of Paris to kill the entrapped insects. When in use, the jar should contain a quantity of loosely crumpled strips of blotting paper, which will increase the efficiency of the cyanide and prevent injury to the trapped insects.

A trap for the study of the movement of insects by wind currents has been designed by Fulton and Chamberlin (1931).

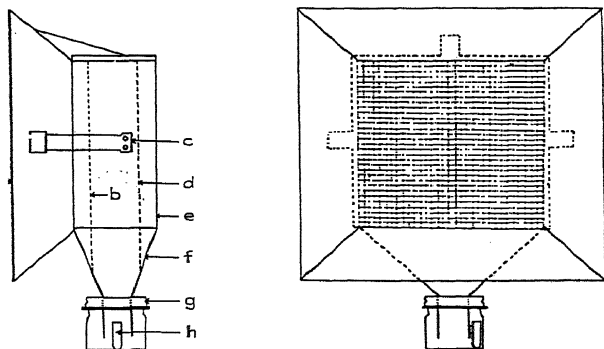


FIG. 238.—A sketch showing the structure of the wind trap of Fulton and Chamberlin. *a*, baffle plate; *b*, hardware cloth $\frac{1}{8}$ inch mesh; *c*, angle brace; *d*, wire screen, 16 mesh; *e*, wire screen, 22 mesh; *f*, funnel; *g*, screw cap for mason jar; *h*, cyanide bag. (After Fulton and Chamberlin.)

In this trap, the insects are blown by the wind through a series of wire screens in which they become entangled, finally falling into a cyanide jar. The trap is installed so that it rotates with the wind, the front of the trap being held facing the wind by a "weather-vane" attachment. The plan of construction is shown in Fig. 238. The trapping of insects larger or smaller than those desired may be avoided to some extent by the proper arrangement of screens of the appropriate size mesh.

MICROBIOLOGICAL METHODS

The nature of insect transmission, *i.e.*, whether mechanical or biological, should be definitely established. If the transmission is biological, the physiological, anatomical, and histological relationships between pathogen and vector should be determined. This will probably involve a study requiring several

different kinds of technique. It will require a study in which the pathogen is cultured from various parts of the insect's body. Insect vectors are usually contaminated with numerous micro-organisms other than the pathogenic one. Many of these will grow on artificial media more rapidly than the pathogen, a fact making it difficult to isolate the pathogen in pure culture or to demonstrate its presence. Differential or selective media are very useful and often necessary if this phase of the investigation is to be successful.

Differential or selective media are those which favor the growth of the organism desired and inhibit the growth of the undesired contaminants. Many different kinds of selective media have been used. One of the simplest of these is made by modifying the acidity reaction. Fungi often may be freed from bacterial contamination by the use of a medium with acidity near or slightly below pH 4.0. This method is based on the fact that most bacteria will not grow well on acid media whereas most fungi are not appreciably inhibited. A 25 per cent solution of lactic acid is commonly used for acidifying the medium.

Fungi and Gram-positive bacteria may be inhibited by adding to the medium a triphenylmethane dye such as crystal violet. Concentrations somewhere between 1 to 50,000 and 1 to 200,000 are usually sufficient to inhibit the Gram-positive species without greatly inhibiting the Gram-negative ones. This medium is particularly helpful in isolating pathogenic bacteria from mixtures of saprophytic ones, for the majority of pathogenic bacteria are Gram-negative whereas the more rapidly growing contaminating bacteria are usually Gram-positive.

It may be necessary to devise a special selective medium to meet the requirements of the particular problem. This can be done only by experimentation. Much time can be saved, however, by utilizing the experience of others, making whatever modifications in known formulae the particular situation demands. In devising a selective medium, the physiological characters of the organism to be cultured and those to be inhibited should be studied with particular reference to tolerance for such things as acidity, bacteriostatic dyes, host extracts, high concentrations of salts, or other special ingredients.

Two examples of selective media helpful in isolating certain bacterial plant pathogens are given here. For additional

formulae of media suitable for specific purposes, see Levine and Schoenlein (1930), Rawlins (1933), Riker and Riker (1936), and the Committee on Bacteriological Technique, Society of American Bacteriologists (1938).

Patel's Agar (Patel 1926).—A selective medium useful in isolating *Phytomonas tumefaciens* and several other bacterial plant pathogens from the soil and other contaminated material.

Distilled water.....	1,000 cc.
Agar.....	15 g.
Sodium taurocholate.....	3 g.
Peptone.....	10 g.
Dextrose.....	20 g.
1 to 1,000 aqueous solution of crystal violet.....	2 cc.

Melt the agar in the water by autoclaving; add the other ingredients, adjust to pH 7, filter, and tube and sterilize in the autoclave.

Ivanoff's Agar (Ivanoff 1933).—A synthetic medium useful in isolating *Phytomonas stewarti* from soil and other contaminated material.

Distilled water.....	1,000 cc.
Agar.....	17.0 g.
Glycerol.....	30.0 cc.
Ferric ammonium citrate.....	10.0 g.
Sodium taurocholate.....	3.0 g.
Sodium chloride.....	15.0 g.
Sodium sulphate.....	2.5 g.
Potassium dibasic phosphate.....	2.5 g.
Calcium chloride.....	1.0 g.
Magnesium sulphate.....	0.1 g.

Add the ingredients to the agar melted in the water, filter, and tube and sterilize in the autoclave.

Pathogenic organisms may be isolated from old and badly contaminated plant or insect material by using the selective action of the host plant. In this method, the host plant is inoculated with the mixed culture taken directly from the contaminated material; if conditions are suitable, infection will occur, forming a fresh lesion from which the pathogen may be isolated by the usual methods of tissue cultures or dilution plates. If the newly formed lesions are of a nature that permits rapid growth of contaminating organisms, it may be necessary to

repeat the process with successive isolations until the selective action of the host has permitted the pathogen to outgrow the contaminating organisms. This method has been used with success by Fulton (1920) in isolating *Phytophthora citri* (Hasse) Bergey *et al.* from the soil and by Leach (1930) in isolating *Erwinia carotovora* from the soil and from the various developmental stages of seed-corn maggot.

In the study of the relationships between pathogen and vector, it may be necessary to dissect out particular organs of the insect. This requires considerable skill and dexterity depending upon the size and nature of the insect. Dissections are usually made under the binocular microscope with the insect secured to a block of wax. Pins may be used in securing the specimen, or it may be partly embedded in the melted wax which is later cooled. If the dissection is made with the view of obtaining cultures of the associated microorganism, it may be desirable to surface-sterilize the insect by immersing it successively in 95 per cent alcohol, a mercuric chloride solution (1 to 1,000) and again in 95 per cent alcohol. The internal organs of insects may dry very rapidly when exposed to dry air and usually become difficult to handle. This trouble may be avoided by covering the specimen with a sterile saline solution.

When pathogens or other microorganisms live symbiotically with their vectors, it is of interest to know whether or not they are transmitted within the egg. The chorions of most insect eggs are relatively impervious to aqueous solutions, and eggs may be surface-sterilized by immersing them in a solution of mercuric chloride (1 to 1,000). The mercuric chloride solution is then removed by washing the eggs in sterile distilled water. The time required for surface sterilization without injury must be determined for each kind of egg. The eggs of the seed-corn maggot and cabbage maggot may be immersed in this solution for at least 2 hours without injury (Leach 1926). This is much longer than is necessary for effective surface sterilization.

HISTOLOGICAL METHODS

Microbiological studies in which the microorganisms are isolated from the insect must be supplemented by histological studies, if the more detailed anatomical relationship between pathogen and vector is to be learned. This usually is accom-

plished best by cutting serial sections from material embedded in paraffin and staining them with a differential stain. Simple smears may be made and stained on glass slides by dissecting out the various organs concerned. This is useful as a rapid method of determining the presence of the microorganism but is not so satisfactory as the paraffin method for more critical studies. Similar histological studies of plant tissue also are frequently necessary in the study of insect transmission of plant diseases. In a study of the mechanism of feeding by insect vectors, both insect and plant tissues must be studied. The techniques for the study of insect and plant histology are too numerous for an extensive treatment in this book. The student should consult the more comprehensive guides to histological technique. Some of the most helpful guides are those published by the following authors: Chamberlain (1932), McClung (1929), Kingsbury and Johannsen (1927), Mallory and Wright (1924), Kennedy (1932), and Becker and Roudabush (1935).

Success will depend to some extent upon the choice of killing fluids and staining formulae. There are numerous formulae from which to choose, and the choice will depend largely upon the material being studied and the worker's preference. The following formulae have proved satisfactory when used by the author for the material indicated.

Fixing Solutions.—The Nawaschin solution is one of the most satisfactory killing and fixing solutions for plant tissues. The solution must be freshly prepared for each lot of material. Fix for 24 hours, and wash in running water for 24 hours.

Nawaschin Solution.

1 % chromic acid solution.....	10.0 cc.
16 % formalin solution.....	4.0 cc.
Glacial acetic acid.....	1.0 cc.

If material must be killed and fixed in the field at the time of collection, the formaldehyde-acetic acid-alcohol solution is satisfactory. No washing in water is necessary, and the material may be kept for some time in the fixing solution without injury.

Formalin-Acetic Acid-Alcohol Solution.

Formalin.....	10.0 cc.
Glacial acetic acid.....	5.0 cc.
Alcohol 50 %.....	200.0 cc.

Zenker's fixing solution is one of the most satisfactory fixing fluids for insect material.

Zenker's Solution.

Solution I

Potassium dichromate.....	2.5 g.
Sodium sulphate.....	1.0 g.
Mercuric chloride.....	5.0 g.
Distilled water.....	100.0 cc.

Solution II

Glacial acetic acid.....	5.0 cc.
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Mix solution I and II just before using. Fix in the solution for 12 to 48 hours, and wash in running water for 12 to 48 hours. Place in 50 per cent alcohol for 3 hours and 70 per cent alcohol for 3 to 24 hours, keeping the material in the dark while in the alcohol solutions. Transfer to 70 per cent alcohol, and add a saturated solution of iodine in 10 per cent alcohol, drop by drop, until the iodine solution is no longer decolorized. The solution may be poured off and replaced with fresh 70 per cent alcohol several times during this process. When iodine treatment is complete, transfer to 80 per cent alcohol and complete the dehydration process.

When it is not convenient to use Zenker's solution or when the insects must be left for a long time in the killing fluid, the formalin-acetic acid-alcohol solution may be used. A slightly modified formula known as Dietrich's solution has been used for several different kinds of insect with excellent results.

Dietrich's Solution.

Alcohol 95 %.....	30.0 cc.
Glacial acetic acid.....	2.0 cc.
Formalin.....	10.0 cc.
Distilled water.....	60.0 cc.

In a study of the mechanics of feeding of insect vectors such as aphids, it is necessary to kill and fix the specimens so that the stylets are not withdrawn from the tissues. A satisfactory method has been described by Dykstra and Whitaker (1938). Leaves and stems on which the insects are feeding are cut from the plant with as little disturbance of the insects as possible.

The entire specimen is dipped in chloroform, removed immediately, and immersed in formalin-acetic-alcohol for 24 hours. The specimens are deaired with an exhaust pump in this solution, then washed in dioxane over calcium chloride for 24 hours. After dehydration, the material is cleared in the following series of dioxane and xylol, leaving it 10 to 15 minutes in each mixture:

1. Xylol 1 part, dioxane 4 parts
2. Xylol 2 parts, dioxane 3 parts
3. Xylol 3 parts, dioxane 2 parts
4. Xylol 4 parts, dioxane 1 part

The material is then removed to pure xylol and embedded in paraffin in the usual manner.

The use of dioxane for dehydration is based on the schedule described earlier by McClung (1936) and by McWhorter and Weier (1936). The substitution of dioxane for alcohol prevents the tissues from becoming brittle, so that ribbon sections can be cut of heavily lignified tissues without disturbing the delicate mouth parts of the insects embedded in the tissue. The method as applied in the above study has been described also by Whitaker (1937). A more complete discussion of the dioxane technique has been published by Mossman (1937), who was largely responsible for introducing the method to American workers.

Staining.—Many different staining formulae are used for demonstrating the presence of bacteria or fungi in plant tissue. One of the more satisfactory of several used by the author is the carbol-fuschin and light-green combination. With this stain, bacteria or fungi are stained a deep red, and cellulose cell walls are stained a bright green. The striking contrast of these colors is of great advantage in locating the microorganisms if they are present in small numbers and allows very sharp differentiation between the pathogen and the tissues of the host plant.

Carbol Fuschin and Light Green.

Fuchsin.....	1.0 g.
Alcohol 95 %.....	10.0 cc.
Carbolic acid crystals.....	5.0 g.
Distilled water.....	100.0 cc.

Stain sections, removed from 10 per cent alcohol, for 3 minutes. Rinse in water, and dehydrate by passing rapidly through alcohol series, being careful not to destain too much. From absolute

alcohol, transfer to a saturated solution of light green in clove oil. Clear by rinsing with clove oil and mount in balsam.

A carbol-fuchsin and picric acid formula recommended by Hertig and Wolbach (1924) has proved satisfactory for demonstrating the presence of bacteria in the body of Dipterous insects (Leach 1931, 1933).

Carbol Fuchsin and Picric Acid.

Solution I

Basic fuchsin.....	0.5 g.
Carbolic acid crystals.....	1.0 g.
Aniline oil.....	0.5 cc.
Alcohol 30 %.....	100.0 cc.

Dissolve fuchsin in alcohol, and add remaining reagents.

Solution II

Picric acid.....	Saturated aqueous solution
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Sections should be steamed over boiling water for 5 minutes in a few drops of solution I poured on the slide. Wash off excess stain in tap water. Decolorize and differentiate by pouring formalin over the sections, a few drops at a time, until the excess color is removed. Rinse in water and counterstain for 1 minute in solution II. Dehydrate quickly in 95 per cent alcohol, absolute alcohol, and xylol. Mount in balsam.

The Gram-Weigert stain (Mallory and Wright 1924) has given excellent results in demonstrating the presence of yeasts, fungus spores, and Gram-positive bacteria in bark beetles and other Coleoptera. It also gives excellent differentiation of the insect tissues. It is not satisfactory if the microorganisms concerned are Gram-negative.

Gram-Weigert Stain (Modified).

Solution I

ALUM HAEMATOKSYLIN

Haematoxylin.....	1.0 g.
Ammonia alum (sat. aq. sol.).....	100.0 cc.
Water.....	300.0 cc.
Potassium permanganate (0.25 % aq. sol.).....	10.0 cc.
A small thymol crystal	

Pulverize haemotoxylin, and dissolve it with the ammonia alum in the water with aid of heat. Cool and add potassium permanganate and then the thymol crystal. The stain is ready for use at once. As the stain ripens, it may be necessary to add a little of the saturated alum solution. Filter each day before using.

Solution II

Eosin..... 5 % aqueous solution

Filter each day before using.

Solution III

ANILINE METHYL VIOLET

Solution A		Solution B
Absolute alcohol.....	33.0 cc.	Saturated aqueous solution of methyl violet
Aniline oil.....	9.0 cc.	
Methyl violet.....	In excess	

Mix 1 part of solution A and 9 parts of solution B not longer than 2 weeks before using. Filter each day before using.

Solution IV

LUGOL'S SOLUTION

Iodine.....	1.0 g.
Potassium iodide.....	2.0 g.
Distilled water.....	100.0 cc.

Stain sections for 3 minutes in alum-haematoxylin solution. Rinse in running water. Stain 3 minutes in 5 per cent eosin solution. Rinse off surplus stain in running water. Stain in solution III (aniline methyl violet) for 5 minutes. Wash 1 minute in running water. Immerse in Lugol's solution for 1 minute. Rinse in running water. Drain off excess water, dry back of slide, and clear in a solution of 1 part xylol and 1 part aniline oil. Agitate slides until excess methyl violet stain is removed. Wash thoroughly in xylol to remove every trace of aniline oil, and mount in balsam.

MISCELLANEOUS TECHNIQUES FOR SPECIAL PURPOSES

Radioactive Substances.—Hamilton (1935) has introduced a novel technique for the study of the course of ingested food in the insect's body in relation to virus transmission. This involves

feeding the insect vectors on a medium containing polonium, a radioactive substance the presence of which can be detected with a fluoroscope. This substance was ingested from agar by *Myzus persicae* and transmitted to the plants on which it subsequently fed in quantities that excluded the possibility of simple mechanical transmission. Recent developments in methods for producing radioactive forms of many of the common elements should provide materials suitable for the more extensive use of this technique in the study of the physiology of insects in relation to virus transmission.

Differential Killing.—When microorganisms survive in the body of an insect through metamorphosis, they are usually reduced to relatively small numbers during certain stages. The microbes do not die, but their growth and multiplication is inhibited by the life processes of the insect. When in this condition they may be difficult to locate by cultural or histological methods. Aid in tracing the microorganisms may be gained by subjecting the insect at the critical stage to subfreezing temperature. The low temperature will usually kill the insect but will not injure the microorganism. If the specimen is then returned to a more moderate temperature, the microorganism, unless it is an obligate parasite, being no longer inhibited by the living tissues of the insect, will begin to grow and can be more easily detected. This method was used successfully by the author (Leach 1933) in tracing the survival of bacteria in the puparia of the seed-corn maggot.

In the study of symbiotic relationships between insects and microorganisms it is often desirable to obtain insects free of their symbiotes. This may present many difficulties. Cleveland in his investigation of the symbiotic relationships between termites and protozoa devised a number of differential treatments by means of which termites were freed of their intestinal protozoa. One method consisted in incubating the termites at 36 degrees centigrade for 24 hours, a treatment that killed the protozoa without injuring the termites. Prolonged starvation of the termites also was often successful. The most satisfactory method, however, consisted in placing the insects in an atmosphere of almost pure oxygen under pressure of 3 or 4 atmospheres for 1 or 2 hours. To what extent these methods can be used with other insects and other microorganisms remains to be determined.

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APPENDIX

In the following appendix, certain facts, pertinent to the subject of insect transmission of plant diseases, have been tabulated for convenient reference. In Table I, the more important insect-transmitted plant diseases have been grouped in one column according to the causal agent, with numerical reference to their respective vectors listed in the opposite column. In Table II, the vectors are grouped in one column, according to order and family, with numerical reference to the diseases transmitted, the latter being given in the opposite column. In Tables III to V, certain representative insect-transmitted plant diseases are compared with respect to significant transmission phenomena. In Table VI, a few representative animal diseases are compared in a similar manner. A study of these tables should aid in giving a better perspective of the subject matter treated in detail in the text.

TABLE I.—SOME OF THE MORE IMPORTANT INSECT-TRANSMITTED PLANT
DISEASES AND THEIR RESPECTIVE INSECT VECTORS

Diseases	Vectors
Diseases caused by toxicogenic insects:	HEMIPTERA
Anasa wilt of cucurbits (1)*	1. <i>Anasa tristis</i> De G.
Froghopper blight of sugar cane (5)	HOMOPTERA
Green spotting of pineapple (2)	2. <i>Pseudococcus brevipes</i> Ckl.
Hopper burn of potato and other plants (3)	3. <i>Empoasca fabae</i> Harris
Mealy-bug wilt of pineapple (2)	4. <i>Paratrioza cockerelli</i> Sulc.
Psyllid yellows of potato (4)	5. <i>Thomaspis saccharina</i> Dist.
Virus diseases:	ORTHOPTERA
A virus disease of rape and rutabaga (6)	1. <i>Melanoplus</i> spp.
Abacá bunchy top (<i>Musa</i> virus 2) (57)	THYSANOPTERA
Abacá mosaic (18)	2. <i>Frankliniella lycopersici</i> Andr.
Alfalfa mosaic (<i>Medicago</i> viruses 1 and 2) (50)	3. <i>Frankliniella moultoni</i> Hood
Aster yellows (<i>Callistephus</i> viruses 1 and 1A) (36, 64, 65)	4. <i>Frankliniella occidentalis</i> Perg
	5. <i>Thrips tabaci</i> Lind.
	HEMIPTERA
	6. <i>Lygus pratensis</i> L.
	7. <i>Piesma cinerea</i> Say

* The numbers in parentheses following the name of the disease refer to the numbers of the insect vectors in the opposite column.

TABLE I.—SOME OF THE MORE IMPORTANT INSECT-TRANSMITTED PLANT DISEASES AND THEIR RESPECTIVE INSECT VECTORS.—(Continued)

Diseases	Vectors
Virus diseases (continued)	8. <i>Piesma quadrata</i> Fieb.
Aucuba mosaic of potato (<i>Solanum virus</i> 9) (54)	HOMOPTERA
Banana bunchy top (<i>Musa virus</i> 1) (57)	9. <i>Aceratagallia sanguinolenta</i> (Provancher)
Banana mosaic (<i>Musa virus</i> 3) (57)	10. <i>Amphorophora rubicola</i> (Oestlund)
Bean mosaic (<i>Phaseolus virus</i> 1) (18, 22, 27, 28, 32, 44, 47, 48, 50, 54, 64)	11. <i>Amphorophora rubi</i> Kalt.
Bean yellow mosaic (<i>Phaseolus virus</i> 2) (48, 50)	12. <i>Amphorophora sensoriata</i> Mason
Beet mosaic (<i>Beta virus</i> 2) (27, 38)	13. <i>Anuraphis padi</i> L. (<i>Aphis pruni</i>)
Blackberry dwarf (<i>Rubus virus</i> 5) (34)	14. <i>Anuraphis tulipae</i> B. de Fonse.
Cabbage ring spot (<i>Brassica virus</i> 1) (54)	15. <i>Aphis apigraveolens</i> Theob.
Cassava mosaic (<i>Manihot virus</i> 1) (29, 30)	16. <i>Aphis convolvuli</i> Kalt.
Cauliflower mosaic (<i>Brassica virus</i> 3) (15, 18, 19, 23, 32, 35, 53, 54, 61, 63)	17. <i>Aphis forbesi</i> Weed.
Celery calico (<i>Apium virus</i> 2) (18)	18. <i>Aphis gossypii</i> Glov.
Common mosaic of pea (<i>Pisum viruses</i> 2 and 2C) (27, 48, 50, 54)	19. <i>Aphis graveolens</i> Essig
Corn mosaic (<i>Zea virus</i> 1) (58)	20. <i>Aphis laburni</i> Kalt. (<i>Aphis leguminosae</i> Theo.)
Cranberry false blossom (<i>Vaccinium virus</i> 1) (42)	21. <i>Aphis maidis</i> Fitch
Cucumber mosaic (<i>Cucumis virus</i> 1) (18, 48, 53, 54, 55, 67, 69)	22. <i>Aphis medicaginis</i> Koch
Curly top of sugar beet (<i>Beta virus</i> 1) (43)	23. <i>Aphis middletonii</i> Thomas
Dahlia mosaic (<i>Dahlia virus</i> 1) (54)	24. <i>Aphis pomi</i> De G.
Enation mosaic of pea (<i>Pisum virus</i> 1) (50)	25. <i>Aphis rhamni</i> Boyer (<i>abbreviata</i> Patch)
Fiji disease of sugar cane (<i>Saccharum virus</i> 2) (59, 60)	26. <i>Aphis rubicola</i> Oestl. (<i>rubiphila</i> Patch)
Freesia mosaic (<i>Freesia virus</i> 1) (16)	27. <i>Aphis rumicis</i> L.
Grape mosaic (<i>Vitis virus</i> 1) (46)	28. <i>Aphis spiraeicola</i> Patch
	29. <i>Bemisia gossypiperda</i> var. <i>mosaicivectura</i> Ghesq.
	30. <i>Bemisia nigeriensis</i> Corb.
	31. <i>Bemisia gossypiperda</i> Misra and Lamba
	32. <i>Brevicoryne brassicae</i> L.
	33. <i>Capitophorus fragaefolii</i> Ckl.
	34. <i>Capitophorus tetrarhodus</i> (Walker)
	35. <i>Cavariella capreae</i> Fabr.
	36. <i>Macrostelus divisa</i> (Uhl.)
	37. <i>Cicadulina mbila</i> Naude
	38. <i>Cicadulina storeyi</i> China
	39. <i>Cicadulina zeae</i> China

TABLE I.—SOME OF THE MORE IMPORTANT INSECT-TRANSMITTED PLANT DISEASES AND THEIR RESPECTIVE INSECT VECTORS.—(Continued)

Diseases	Vectors
Virus Diseases (continued)	
Henbane mosaic (<i>Hyoscyamus</i> virus 1) (54)	40. <i>Delphacodes striatella</i> Fall. (<i>Liburnia striatella</i> Fall.)
Iris mosaic (<i>Iris</i> virus 1) (48, 54)	41. <i>Euscelis eritiosus</i> (Uhler)
Leaf curl of cotton (<i>Gossypium</i> virus 1) (31)	42. <i>Euscelis striatulus</i> Fall.
Leaf curl of sugar beet (<i>Beta</i> virus 3) (8)	43. <i>Eutettix tenellus</i> Baker
Leaf curl of tobacco (<i>Nicotiana</i> virus 10) (31)	44. <i>Hyalopterus atriplicis</i> (L.)
Lettuce mosaic (<i>Lactuca</i> virus 1) (51, 54)	45. <i>Hysteronura setariae</i> Thomas
Little peach (<i>Prunus</i> virus 1A) (52)	46. <i>Lecanium corni</i> Bouché
Mosaic of crucifers (<i>Brassica</i> virus 4) (32, 54)	47. <i>Macrosiphum ambrosiae</i> (Thomas)
Mottle of corn (<i>Zea</i> virus 3) (37, 38, 39)	48. <i>Macrosiphum gei</i> Koch (<i>solanifolii</i>) Ashm.
Peach mosaic (<i>Prunus</i> virus 5) (13)	49. <i>Macrosiphum pelargonii</i> Kalt.
Peach yellows (<i>Prunus</i> virus 1) (52)	50. <i>Macrosiphum (Illinoia) pisi</i> Kalt.
Peanut rosette (<i>Arachis</i> virus 1) (20)	51. <i>Macrosiphum sonchi</i> L.
Pelargonium leaf curl (<i>Pelargonium</i> virus 1) (49)	52. <i>Macropsis trimaculata</i> Fitch
Pineapple yellow spot (<i>Ananas</i> virus 1) (5)	53. <i>Myzus circumflexus</i> Buckt.
Potato leaf roll (<i>Solanum</i> virus 14) (27, 53, 54, 55)	54. <i>Myzus persicae</i> Sulz.
Potato mosaic (<i>Solanum</i> virus 3) (25)	55. <i>Myzus pseudosolani</i> Theob.
Potato spindle tuber (<i>Solanum</i> virus 12) (1, 6, 48, 54, 69, 70, 71, 72)	56. <i>Nephotettix apicalis</i> var. <i>cincticeps</i> Motsch.
Potato streak (leaf drop) (<i>Solanum</i> viruses 2 and 3) (54)	57. <i>Pentalonia nigronervosa</i> Coq.
Potato tuber blotch (<i>Solanum</i> virus 8) (54)	58. <i>Peregrinus maidis</i> Ashm.
Potato unmottled curly dwarf (1, 6, 41, 48, 54, 69, 70, 71, 72)	59. <i>Perkinsiella saccharicida</i> Kirk.
Potato yellow dwarf (<i>Solanum</i> virus 16) (9, 48, 54)	60. <i>Perkinsiella vastatrix</i> Breddin
Rape Savoy (6)	61. <i>Rhopalosiphum melliferum</i> Hottes
Raspberry leaf curl (<i>Rubus</i> viruses 3 and 3A) (26)	62. <i>Rhopalosiphum prunifoliae</i> Fitch
	63. <i>Rhopalosiphum pseudobrasicae</i> Davis
	64. <i>Thamnotettix geminatus</i> Van D.
	65. <i>Thamnotettix montanus</i> Van D.
	66. <i>Deltocephalus dorsalis</i> Motsch.
	COLEOPTERA
	67. <i>Diabrotica duodecimpunctata</i> Oliv.
	68. <i>Diabrotica vittata</i> Fabr.
	69. <i>Disonycha triangularis</i> (Say)
	70. <i>Epitrix cucumeris</i> Harris
	71. <i>Leptinotarsa decimlineata</i> Say
	72. <i>Systena taeniata</i> (Say)

TABLE I.—SOME OF THE MORE IMPORTANT INSECT-TRANSMITTED PLANT DISEASES AND THEIR RESPECTIVE INSECT VECTORS.—(Continued)

Diseases	Vectors
Virus diseases (continued)	
Raspberry yellow mosaic (Rubus virus 2) (10, 11, 12)	<i>(See preceding pages)</i>
Red raspberry mosaic (Rubus virus 1) (10, 11, 12)	
Rice dwarf (Oryza virus 1) (56, 66)	
Savoy of sugar beet (Beta virus 5) (7)	
Spotted wilt (Lycopersicum virus 3) (2, 3, 4, 5)	
Stock mosaic (Matthiola virus 1) (32, 54, 63)	
Strawberry crinkle (Fragaria virus 2) (33)	
Strawberry dwarf (Fragaria virus 4) (17)	
Strawberry witches'-broom (Fragaria virus 3) (33)	
Strawberry xanthosis (Fragaria virus 1) (33)	
Streak of corn (Zea virus 2) (37, 38, 39)	
Stripe of rice (Oryza virus 2) (40)	
Sugar-beet mosaic (Beta virus 2) (54, 27)	
Sugar-beet yellows (Beta virus 4) (27, 54)	
Sugar-cane mosaic (Saccharum virus 1) (21, 45)	
Tobacco mosaic (Nicotiana virus 1) (48, 53, 55)	
Tulip mosaic (Tulipa virus 1) (14, 48, 49, 54)	
Turnip mosaic (Brassica virus 2) (32)	
White-clover mosaic (Trifolium virus 1) (27, 50)	
Yellow dwarf of onion (Allium virus 1) (11, 18, 21, 24, 27, 32, 44, 45, 48, 50, 54, 62, 63)	
Yellow flat of lily (Lilium virus 1) (18)	

TABLE I.—SOME OF THE MORE IMPORTANT INSECT-TRANSMITTED PLANT DISEASES AND THEIR RESPECTIVE INSECT VECTORS.—(Continued)

Diseases	Vectors
Bacterial diseases	THYSANOPTERA
Bacterial disease of willows (<i>Pseudomonas saliciperda</i>) (25)	1. <i>Heliothrips femoralis</i> Reut.
Bacterial gall of Douglas fir (<i>Bacterium pseudosugae</i>) (9)	HEMIPTERA
Bacterial rot of apples (<i>Phytomonas melophthora</i>) (37)	2. <i>Adelphocoris rapidus</i> (Say)
Bacterial wilt of corn (Stewart's disease) (<i>Phytomonas stewarti</i>) (13, 14, 15, 16, 17)	3. <i>Campylomma verbasci</i> (Meyer)
Bacterial wilt of cucurbits (<i>Erwinia tracheiphila</i>) (16, 19)	4. <i>Lygus pratensis</i> L.
Bacterial wilt of Solanaceae (<i>Erwinia solanacearum</i>) (20)	5. <i>Orthotylus flavosparvus</i> (Sahlb.)
Bacteriosis of the prickly-pear plant (<i>Erwinia cacticida</i>) (28, 29, 30, 31, 32)	6. <i>Plagiognathus politus</i> Uhl.
Bean bacteriosis (<i>Phytomonas medicaginis</i> var. <i>phaseolicola</i>) (1)	7. <i>Poecilocytus basalis</i> Reut.
Black rot of crucifers (<i>Phytomonas campestris</i>) (34)	HOMOPTERA
Fire blight (<i>Erwinia amylovora</i>) (2, 3, 4, 5, 6, 7, 8, 10, 11, 12, 18, 21, 22, 23, 24, 26, 27, 33, 35, 38, 41, 44, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56)	8. <i>Empoasca mali</i> Le Baron
Olive knot (<i>Erwinia savastanoi</i>) (36)	9. <i>Chermes cooleyi</i> Gill
Potato blackleg (<i>Erwinia carotovora</i>) (43, 45)	10. <i>Aphis avenae</i> Fay
Soft rot of crucifers (<i>Erwinia carotovora</i>) (42)	11. <i>Aphis pomi</i> De Geer
The heart rot of celery (<i>Erwinia carotovora</i>) (39, 40)	COLEOPTERA
	12. <i>Hippodamia convergens</i> Guer.
	13. <i>Phyllophaga</i> sp.
	14. <i>Chaetocnema denticulata</i> Ill.
	15. <i>Chaetocnema pulicaria</i> Melsh.
	16. <i>Diabrotica duodecimpunctata</i> Oliv.
	17. <i>Diabrotica longicornis</i> Say
	18. <i>Diabrotica soror</i> Le C.
	19. <i>Diabrotica vittata</i> Fabr.
	20. <i>Leptinotarsa decimlineata</i> Say
	21. <i>Orsodacne atra</i> (Ahr.)
	22. <i>Attagenus piceus</i> Oliv.
	23. <i>Anthrenus</i> sp.
	24. <i>Glischrochilus fasciatus</i> Oliv.
	25. <i>Cryptorrhynchus lapathi</i> L.
	26. <i>Melanotus oregonensis</i> (Le C.)
	27. <i>Scolytus rugulosus</i> Ratz.
	LEPIDOPTERA
	28. <i>Mimorista flavidissimalis</i> Grote.
	29. <i>Cactoblastis beutyus</i> Dyar
	30. <i>Cactoblastis cactorum</i> Berg.
	31. <i>Melipotera prodentalis</i> Walker
	32. <i>Melipotera junctelinsella</i> Hulst.
	33. <i>Carpocapsa pomonella</i> L.
	34. <i>Plusia brassicae</i> Riley
	DIPTERA
	35. <i>Bibio albipennis</i> Say

TABLE I.—SOME OF THE MORE IMPORTANT INSECT-TRANSMITTED PLANT DISEASES AND THEIR RESPECTIVE INSECT VECTORS.—(Continued)

Diseases

Vectors

36. *Dacus olea* Rossi
37. *Rhagoletis pomonella* Walsh
38. *Drosophila funebris* Fabr.
39. *Elachiptera costata* Leow.
40. *Scaptomyza graminum* Fall.
41. *Hylemyia antiqua* Meig.
42. *Hylemyia brassicae* Bouché
43. *Hylemyia cilicrura* Rond.
44. *Hylemyia lipsia* Walker
45. *Hylemyia trichodactyla* Rond.
46. *Pegomyia calypttrala* Zett.
47. *Cynomyia cadaverina* Desv.
48. *Musca domestica* L.
49. *Muscina assimilis* Fall.
50. *Muscina stabulans* Fall.

HYMENOPTERA

51. *Formica fusca* L. var. *subsericea* Say
52. *Formica pallidefulva* Latr. subsp. *schaufussi* Mayr. var. *incerta* Emery
53. *Lasius niger* (L.) var. *americanus* Emery
54. *Prenolepis imparis* Say
55. *Polistes* sp.
56. *Vespula* sp.

Fungus diseases

- Blackleg of cabbage (*Phoma lingam*) (46)
- Blue stain of coniferous trees (*Ceratostomella* spp.) (34, 35, 36, 36a, 36b, 36c)
- Blue stain of spruce (*Ceratostomella piceaperda*) (34a)
- Blue stain of Douglas fir (*Ceratostomella pseudotsugae*) (34b)
- Brown stain of white fir (*Trichosporium symbioticum*) (42b)
- Brown stain of white fir (*Spicaria anomala*) (42c)
- Canker of apple (*Diplodia griffoni*) (26)

ORTHOPTERA

1. *Oecanthus angustipennis* Fitch
2. *Oecanthus niveus* De G.
3. *Chortophaga viridifasciata* var. *austrator* De G.
4. *Dissosteira carolina* L.
5. *Encoptolophus texensis* Brun.
6. *Melanoplus differentialis* Thomas
7. *Melanoplus femur-rubrum* De G.
8. *Melanoplus mexicanus* Sauss.
9. *Schistocerca americana* Drury
10. *Schistocerca obscura* Fabr.
11. *Spharagemon cristatum* Seudd.
12. *Tomonotus aztecus* Sauss.
13. *Trimerotropis citrina* Seudd.

TABLE I.—SOME OF THE MORE IMPORTANT INSECT-TRANSMITTED PLANT DISEASES AND THEIR RESPECTIVE INSECT VECTORS.—(Continued)

Diseases	Vectors
Fungus diseases (continued)	HEMIPTERA
Chestnut blight (<i>Endothia parasitica</i>) (32, 33)	14. <i>Dysdercus cingulatus</i> (Fabricius)
Cotton wilt (<i>Fusarium vasinfectum</i> Atk.) (3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13)	15. <i>Dysdercus fasciatus</i> Signoret
Downy mildew of lima beans (<i>Phytophthora phaseoli</i> Thaxter) (48)	16. <i>Dysdercus intermedius</i> Distant
Endosepsis of figs (<i>Fusarium moniliforme</i> var. <i>fici</i>) (47)	17. <i>Dysdercus nigrofasciatus</i> Stål.
Ergot of cereals and grasses (<i>Claviceps paspali</i>) (31, 44)	18. <i>Dysdercus supersticiosus</i> (Fabr.)
European canker (<i>Nectria galligena</i>) (26)	19. <i>Dysdercus</i> spp.
Inspissosis of citrus fruits (<i>Nematospora coryli</i>) (21, 24)	20. <i>Leptoglossus balteatus</i> (L.)
Perennial canker of apple (<i>Gloeosporium perennans</i>) (26)	21. <i>Leptoglossus zonatus</i> (Dall.)
Smut of figs (<i>Aspergillus niger</i>) (30, 45)	22. <i>Phthia picta</i> (Drury)
Sooty mold of orange (<i>Capnodium citri</i> and other fungi) (25, 27, 28, 29)	23. <i>Nezara hilaris</i> Say
Souring of figs (yeast) (30, 45)	24. <i>Nezara viridula</i> L.
Stigmatomycosis (<i>Nematospora</i> spp.) (14, 15, 16, 17, 18, 19, 20, 22, 23, 24, 24a)	24a. <i>Antestia lineaticollis</i> (Stål.)
Dutch elm disease (<i>Ceratostomella ulmi</i>) (37, 38, 39, 40, 41, 42, 43)	HOMOPTERA
Tree cricket canker of apple (<i>Leptosphaeria coniothyrium</i>) (1, 2)	25. <i>Aphis gossypii</i> Glov.
"Yeast spot" of lima beans (<i>Nematospora phaseoli</i>) (23)	26. <i>Schizoneura lanigera</i> Hausmann
Potato scab (<i>Actinomyces scabies</i>) (31a)	27. <i>Aleyrodes citri</i> R. and H.
Plum wilt (<i>Lasioidiplodia triflorae</i>) (43a)	28. <i>Ceroplastes floridensis</i> Comstock
Leaf spot of tomatoe (<i>Septoria lycopersici</i>) (31a)	29. <i>Dactylodius citri</i> Risso
	COLEOPTERA
	30. <i>Carpophilus hemipterus</i> L.
	31. <i>Carabidae</i> (species not known)
	31a. <i>Epitrix cucumeris</i> Harris
	32. <i>Leptostylus macula</i> Say
	33. <i>Leptura nitens</i> Forst. = <i>Strophiona nitens</i> Forst.
	34. <i>Dendroctonus ponderosae</i> Hopk.
	34a. <i>Dendroctonus piceaperda</i>
	34b. <i>Dendroctonus pseudosugae</i>
	35. <i>Ips grandicollis</i> Eich.
	36. <i>Ips pini</i> Say
	36a. <i>Ips emarginatus</i> (Le C.)
	36b. <i>Ips interger</i> (Eich.)
	36c. <i>Ips oregoni</i> (Eich.)
	37. <i>Scolytus scolytus</i> Fabr.
	38. <i>Scolytus multistriatus</i> Marsh.
	39. <i>Scolytus sulcifrons</i> Rey.
	40. <i>Scolytus affinis</i> Eggers
	41. <i>Scolytus laevis</i> Chapuis

TABLE I.—SOME OF THE MORE IMPORTANT INSECT-TRANSMITTED PLANT DISEASES AND THEIR RESPECTIVE INSECT VECTORS.—(Continued)

Diseases	Vectors
	42. <i>Scolytus pygmaeus</i> Fabr.
	42a. <i>Scolytus sulcatus</i> Le C.
	42b. <i>Scolytus ventralis</i> Le C.
	42c. <i>Scolytus praeceps</i> Le C.
	43. <i>Hylurgopinus rufipes</i> Eich.
	LEPIDOPTERA
	43a. <i>Aegeria exitosa</i> Say
	DIPTERA
	44. <i>Sciara thomae</i> L.
	45. <i>Drosophila ampelophila</i> Leow.
	46. <i>Hylemyia brassicae</i> Bouché
	HYMENOPTERA
	47. <i>Blastophaga psenes</i> L.
	48. Bees
Protozoan diseases	HEMIPTERA
Flagellosis of laticiferous plants	1. <i>Dieuches humilis</i> Reut.
<i>Leptomonas davidi</i> (1, 3, 6)	2. <i>Lygaeus kalmii</i> Stål.
<i>Herpetomonas elmassiani</i> (4)	3. <i>Nysius euphorbiae</i> Horvath
<i>Herpetomonas lygaeorum</i> (2)	4. <i>Oncopeltus fasciatus</i> Dallas
<i>Phytomonas leptovisorum</i> (5)	5. <i>Lincus securiger</i> (Bredin)
	6. <i>Stenocephalus agilis</i> Scopoli

TABLE II.—SOME OF THE MORE IMPORTANT VECTORS OF PLANT DISEASES AND THE DISEASES THAT THEY TRANSMIT

Vectors	Diseases
ORTHOPTERA	
Gryllidae:	
<i>Oecanthus niveus</i> De G. (1)*	1. Tree-cricket canker of apple (<i>Leptosphaeria coniothyrium</i>)
<i>Oecanthus angustipennis</i> Fitch (1)	2. Potato spindle tuber (<i>Solanum virus 12</i>)
Locustidae:	3. Potato unmottled curly dwarf (<i>Solanum virus 13</i>)
<i>Chortophaga viridifasciata</i> var. <i>austrator</i> De G. (4)	4. Cotton wilt (<i>Fusarium vasinfectum</i>)
<i>Dissosteira carolina</i> L. (4)	
<i>Encoptolophus texensis</i> Brun. (4)	
<i>Melanoplus differentialis</i> Thomas (4)	
<i>Melanoplus femur-rubrum</i> De G. (4)	
<i>Melanoplus mexicanus</i> Sauss. (4)	
<i>Melanoplus</i> spp. (2, 3)	

* The numbers in parentheses following the name of the vector refer to the numbers of the diseases transmitted as given in the opposite column.

TABLE II.—SOME OF THE MORE IMPORTANT VECTORS OF PLANT DISEASES AND THE DISEASES THAT THEY TRANSMIT.—(Continued)

Vectors	Diseases
<i>Schistocerca americana</i> Drury (4)	
<i>Schistocerca obscura</i> Fabr. (4)	
<i>Spharagemon cristatum</i> Seudd. (4)	
<i>Tomonotus aztecus</i> Sauss. (4)	
<i>Trimerotropis citrina</i> Seudd. (4)	
THYSANOPTERA	
Thripidae:	
<i>Frankliniella lycopersici</i> Andr. (2)	1. Pineapple yellow spot (<i>Ananas</i> virus 1)
<i>Frankliniella moultoni</i> Hood (2)	2. Tomato spotted wilt (<i>Lyco-</i> <i>persicum</i> virus 3)
<i>Frankliniella occidentalis</i> Perg. (2)	3. Bean bacteriosis (<i>Phytomonas</i> <i>medicaginis</i> var. <i>phaseolicola</i>)
<i>Heliothrips femoralis</i> Reut. (3)	
<i>Thrips tabaci</i> Lind. (1, 2)	
HEMIPTERA	
Lygaeidae:	
<i>Dieuches humilis</i> Reut. (1a)	1. Flagellosis of lacticiferous plants
<i>Lygaeus kalmii</i> Stål. (1c)	a. <i>Leptomonas davidi</i>
<i>Nysius euphorbiae</i> Horvath (1a)	b. <i>Herpetomonas elmassiani</i>
<i>Oncopeltus fasciatus</i> Dallas (1b)	c. <i>Herpetomonas lygaeorum</i>
Miridae:	
<i>Adelphocoris rapidus</i> (Say) (5)	2. Potatospindle tuber (<i>Solanum</i> virus 12)
<i>Campylomma verbasci</i> (Meyer) (5)	3. Potato unmottled curly dwarf (<i>Solanum</i> virus 13)
<i>Lygus pratensis</i> L. (2, 3, 4, 5, 6)	4. A virus disease of rape and rutabaga
<i>Orthotylus flavosparsus</i> (Sahlb.) (5)	5. Fire blight (<i>Erwinia amylovora</i>)
<i>Plagiognathus politus</i> Uhl. (5)	6. Leaf curl of sugar beet (<i>Beta</i> virus 3)
<i>Peciloscytus basilis</i> Reut. (5)	6a. Rape savoy
Tingidae:	
<i>Piesma cinerea</i> (Say) (7)	7. Savoy of sugar beet (<i>Beta</i> virus 5)
<i>Piesma quadrata</i> Fieb. (6a)	8. Anasa wilt of cucurbits
Coreidae:	
<i>Anasa tristis</i> De G. (8)	9. Stigmatomycosis (<i>Nemato-</i> <i>spora</i> spp.)
<i>Dysdercus cingulatus</i> (Fabr.) (9)	10. Inspissosis of citrus fruits (<i>Nematospora coryli</i>)
<i>Dysdercus fasciatus</i> Signaret (9)	11. "Yeast spot" of lima beans (<i>Nematospora phaseoli</i> Win-
<i>Dysdercus intermedius</i> Distant (9)	gard)
<i>Dysdercus nigrofasciatus</i> Stål. (9)	

TABLE II.—SOME OF THE MORE IMPORTANT VECTORS OF PLANT DISEASES AND THE DISEASES THAT THEY TRANSMIT.—(Continued)

Vectors	Diseases
<i>Dysdercus supersticiosus</i> (Fabr.) (9)	12. Phloem necrosis of coffee (<i>Phytophthora leptovascularum</i>)
<i>Anesthia lineaticollis</i> Stål. (9)	
<i>Dysdercus</i> spp. (9)	
<i>Leptoglossus bolitatus</i> (Linné) (9)	
<i>Leptoglossus zonatus</i> (Dall.) (10)	
<i>Phthia picta</i> (Drury) (9)	
<i>Stenocephalus agilis</i> Scopoli (1a)	
Pentatomidae:	
<i>Lincus spathuliger</i> (Breddin) (12)	
<i>Nezara hiliaris</i> Say (11)	
<i>Nezara viridula</i> L. (9, 10)	
HOMOPTERA	
Cicadellidae:	
<i>Aceratagalliasanguinolenta</i> (Provancher) (11)	1. Hopper burn of potato and other plants
<i>Cicadulina (Balclutha) mbila</i> Naude (8, 13)	2. Aster yellows (<i>Callistophorus</i> viruses 1 and 1A)
<i>Cicadulina storeyi</i> China (8, 13)	3. Corn mosaic (<i>Zea virus</i> 1)
<i>Cicadulina zeei</i> China (8, 13)	4. Cranberry false blossom (<i>Vaccinium virus</i> 1)
<i>Deltocephalus dorsalis</i> Motsch. (12)	5. Curly top of sugar beet (<i>Beta virus</i> 1)
<i>Empoasca fabae</i> Harris (1)	6. Fiji disease of sugar cane (<i>Saccharum virus</i> 2)
<i>Empoasca mali</i> Le Baron (14)	7. Little peach (<i>Prunus virus</i> 1A)
<i>Euscelis eximius</i> (Uhl.) (10)	8. Mottle of corn (<i>Zea virus</i> 3)
<i>Euscelis striatulus</i> Fall. (4)	9. Peach yellows (<i>Prunus virus</i> 1)
<i>Eutettix tenellus</i> Baker (5)	10. Potato unmottled curly dwarf (<i>Solanum virus</i> 13)
<i>Macrostes divisus</i> (Uhl.) (2)	11. Potato yellow dwarf (<i>Solanum virus</i> 16)
<i>Macropsis trimaculata</i> Fitch (7, 9)	12. Rice dwarf (<i>Oryza virus</i> 1)
<i>Nephotettix apicalis</i> var. <i>cincticeps</i> Motsch. (12)	13. Streak of corn (<i>Zea virus</i> 2)
<i>Thamnotettix geminatus</i> Van D. (2)	14. Fire blight (<i>Erwinia amylovora</i>)
<i>Thamnotettix montanus</i> Van D. (2)	15. Stripe of rice (<i>Oryza virus</i> 2)
Fulgoridae:	16. Psyllid yellows of potato
Delphacinae:	17. Bacterial gall of Douglas fir (<i>Bacterium pseudotsugae</i>)
<i>Delphacodes striatella</i> Fall. (15)	18. Abacá bunchy top (<i>Musa virus</i> 2)
<i>Liburnia striatella</i> Fall. (15)	
<i>Peregrinus maidis</i> Ashm. (3)	

TABLE II.—SOME OF THE MORE IMPORTANT VECTORS OF PLANT DISEASES AND THE DISEASES THAT THEY TRANSMIT.—(Continued)

Vectors	Diseases
<i>Perkinsiella saccharicida</i> Kirk. (6)	19. Alfalfa mosaic (Medicago viruses 1 and 2)
<i>Perkinsiella vastatrix</i> Breddin (6)	20. Aucuba mosaic of potato (Solanum virus 9)
Chermidae:	21. Banana bunchy top (Musa virus 1)
<i>Chermes cooleyi</i> Gill (17)	22. Banana mosaic (Musa virus 3)
<i>Paratrioza cockerelli</i> Sulc. (16)	23. Bean mosaic (Phaseolus virus 1)
Aphididae:	24. Bean yellow mosaic (Phaseolus virus 2)
<i>Amphorophora rubicola</i> (Oestlund) (50, 51)	25. Beet mosaic (Beta virus 2)
<i>Amphorophora rubi</i> Kalt. (50, 51, 63)	26. Blackberry dwarf (Rubus virus 5)
<i>Amphorophora sensoriata</i> Mason (50, 51)	27. Cabbage ring spot (Brassica virus 1)
<i>Anuraphis padi</i> L. (<i>Aphis pruni</i>) (39)	28. Cauliflower mosaic (Brassica virus 3)
<i>Anuraphis tulipae</i> B. de Fonsc. (60)	29. Celery calico (Apium virus 2)
<i>Aphis apigraveolens</i> Theob. (28)	30. Common mosaic of pea (Pisum viruses 2 and 2C)
<i>Aphis avenae</i> Fabr. (65)	31. Cucumber mosaic (Cucumis virus 1)
<i>Aphis convolvuli</i> Kalt. (34)	32. Dahlia mosaic (Dahlia virus 1)
<i>Aphis forbesi</i> Weed (54)	33. Enation mosaic of pea (Pisum virus 1)
<i>Aphis gossypii</i> Glov. (23, 28, 29, 31, 63, 64, 74, 78)	34. Freesia mosaic (Freesia virus 1)
<i>Aphis graveolens</i> Essig (28)	35. Henbane mosaic (Hyoscyamus virus 1)
<i>Aphis laburni</i> Kalt. (<i>Aphis leguminosae</i> Theo.) (40)	36. Iris mosaic (Iris virus 1)
<i>Aphis maidis</i> Fitch (58, 63)	37. Lettuce mosaic (Lactuca virus 1)
<i>Aphis medicaginis</i> Koch (23)	38. Mosaic of crucifers (Brassica virus 4)
<i>Aphis middletonii</i> Thomas (28)	39. Peach mosaic (Prunus virus 5)
<i>Aphis pomi</i> De G. (63, 65)	40. Peanut rosette (Arachis virus 1)
<i>Aphis rhamni abbreviata</i> Patch (43)	41. Pelargonium leaf curl (Pelargonium virus 1)
<i>Aphis rubicola</i> Oestl. (<i>rubiphila</i> Patch) (49)	42. Potato leaf roll (Solanum virus 14)
<i>Aphis rumicis</i> L. (23, 25, 30, 42, 57, 62, 63, 70)	
<i>Aphis spiraeicola</i> Patch (23)	
<i>Brevicoryne brassicae</i> L. (23, 28, 38, 52, 61, 63)	
<i>Capitophorus fragaefolii</i> Ckl. (53, 55, 56)	
<i>Capitophorus tetraerhodus</i> (Walker) (26)	

TABLE II.—SOME OF THE MORE IMPORTANT VECTORS OF PLANT DISEASES AND THE DISEASES THAT THEY TRANSMIT.—(Continued)

Vectors	Diseases
<i>Cavariella capreae</i> Fabr. (28)	43. Potato mosaic (<i>Solanum virus</i> 3)
<i>Hyalopterus atriplicis</i> (L.) (23, 65)	44. Potato spindle tuber (<i>Solanum virus</i> 12)
<i>Hysteronura setariae</i> Thomas (58, 63)	45. Potato streak (leaf drop) <i>Solanum viruses</i> 2 and 3)
<i>Macrosiphum ambrosiae</i> (Thomas) (23)	46. Potato tuber blotch (<i>Solanum virus</i> 8)
<i>Macrosiphum gei</i> Koch (solani-folii Ashm.) (23, 24, 25, 30, 31, 36, 44, 47, 59, 60, 63)	47. Potato unmottled curly dwarf (<i>Solanum virus</i> 13)
<i>Macrosiphum pelargonii</i> Kalt. (41, 60)	48. Potato yellow dwarf (<i>Solanum virus</i> 16)
<i>Macrosiphum (Illinoia) pisi</i> (Kalt.) (19, 23, 24, 30, 33, 62, 63)	49. Raspberry leaf curl (<i>Rubus viruses</i> 3 and 3A)
<i>Macrosiphum sonchi</i> L. (37)	50. Raspberry yellow mosaic (<i>Rubus virus</i> 2)
<i>Myzus circumflexus</i> Buckt. (28, 31, 42, 59)	51. Red-raspberry mosaic (<i>Rubus virus</i> 1)
<i>Myzus persicae</i> Sulz. (20, 23, 27, 28, 30, 31, 32, 35, 36, 37, 38, 42, 44, 45, 46, 47, 48, 52, 57, 60, 63, 70)	52. Stock mosaic (<i>Matthiola virus</i> 1)
<i>Myzus pseudosolani</i> Theob. (31, 42, 59)	53. Strawberry crinkle (<i>Fragaria virus</i> 2)
<i>Pentalonia nigronervosa</i> Coq. (18, 21, 22)	54. Strawberry dwarf (<i>Fragaria virus</i> 4)
<i>Rhopalosiphum melliferum</i> Hottes (28)	55. Strawberry witches'-broom (<i>Fragaria virus</i> 3)
<i>Rhopalosiphum prunifoliae</i> Fitch (63)	56. Strawberry xanthosis (<i>Fragaria virus</i> 1)
<i>Rhopalosiphum pseudobrassicae</i> Davis (23, 28, 52, 63)	57. Sugar-beet yellows (<i>Beta virus</i> 4)
<i>Schizoneura lanigera</i> Hausman (66, 67, 68)	58. Sugar-cane mosaic (<i>Saccharum virus</i> 1)
Aleyrodidae:	59. Tobacco mosaic (<i>Nicotiana virus</i> 1)
<i>Aleyrodes citri</i> R. and H. (74)	60. Tulip mosaic (<i>Tulipa virus</i> 1)
<i>Bemisia gossypiperda</i> var. <i>mosaicivectura</i> Ghesq. (71)	61. Turnip mosaic (<i>Brassica virus</i> 2)
<i>Bemisia nigeriensis</i> Corb. (71)	62. White-clover mosaic (<i>Trifolium virus</i> 1)
<i>Bemisia gossypiperda</i> Misra and Lamba (72, 73)	63. Yellow dwarf of onion (<i>Allium virus</i> 1)
Coccidae:	64. Yellow flat of lily (<i>Lilium virus</i> 1)
<i>Pseudococcus brevipes</i> (Ckl.) (75, 76)	

TABLE II.—SOME OF THE MORE IMPORTANT VECTORS OF PLANT DISEASES AND THE DISEASES THAT THEY TRANSMIT.—(Continued)

Vectors	Diseases
<i>Ceroplastes floridensis</i> Comstock (74)	65. Fire blight (<i>Erwinia amylovora</i>)
<i>Dactylodius citri</i> Risso (74)	66. Canker of apple (<i>Diplodia griffora</i>)
<i>Lecanium corni</i> Bouché (77)	67. European canker (<i>Nectria galligena</i>)
	68. Perennial canker of apple (<i>Gloeosporium perennans</i>)
	70. Sugar-beet mosaic (Beta virus 2)
	71. Cassava mosaic (Manihot virus 1)
	72. Leaf curl of cotton (Gossypium virus 1)
	73. Leaf curl of tobacco (Nicotiana virus 10)
	74. Sooty mold of orange (<i>Capnodium citri</i>)
	75. Green spotting of pineapple
	76. Mealy-bug wilt of pineapple
	77. Grape mosaic (Vitis virus 1)
	78. Abacá mosaic
	79. Froghopper blight of sugar
COLEOPTERA	
Nitidulidae:	1. Souring of figs (yeast)
<i>Carpophilus hemipterus</i> L. (1, 2)	2. Smut of figs (<i>Aspergillus niger</i>)
Coccinellidae:	3. Fire blight (<i>Erwinia amylovora</i>)
<i>Hippodamia convergens</i> Guer. (3)	4. Ergot of cereals and grasses (<i>Claviceps paspali</i>)
Carabidae:	5. Bacterial wilt of corn (<i>Phytophthora stewarti</i>)
Unidentified species (4)	6. Chestnut blight (<i>Endothia parasitica</i>)
Scarabaeidae:	7. Cucumber mosaic (Cucumis virus 1)
<i>Phyllophaga</i> sp. (5)	8. Potato spindle tuber (Solanum virus 12)
Cerambycidae:	9. Potato unmottled curly dwarf (Solanum virus 13)
<i>Leptostylus macula</i> Say = <i>Astylopsis macula</i> (Say) (6)	10. Bacterial wilt of cucurbits (<i>Erwinia tracheiphila</i>)
<i>Leptura nitens</i> Forst. = <i>Strophiona nitens</i> (Forst.) (6)	11. Bacterial wilt of Solanaceae (<i>Erwinia solanacearum</i>)
Chrysomelidae:	
<i>Chaetocnema denticulata</i> Ill. (5)	
<i>Chaetocnema pulicaria</i> Melsh. (5)	

TABLE II.—SOME OF THE MORE IMPORTANT VECTORS OF PLANT DISEASES AND THE DISEASES THAT THEY TRANSMIT.—(Continued)

Vectors	Diseases
<i>Diabrotica duodecimpunctata</i> Oliv. (5, 7, 10)	12. Bacterial disease of willows [<i>Pseudomonas</i> (<i>Phytomonas</i>) <i>saliciperda</i>]
<i>Diabrotica longicornis</i> Say (5)	13. Blue stain of coniferous trees (<i>Graphium</i> spp., <i>Ceratostomella</i> spp.)
<i>Diabrotica soror</i> Le C. (3)	14. The Dutch elm disease [<i>Ceratostomella ulmi</i> (Schw.) Buisman]
<i>Diabrotica vittata</i> Fab. (7, 10)	15. Potato scab (<i>Actinomyces scabies</i>)
<i>Disonycha triangularis</i> (Say) (8)	16. Brown stain of white fir (<i>Trichosporium symbioticum</i>)
<i>Epitrix cucumeris</i> Harris (8, 9, 15, 20)	17. Brown stain of white fir (<i>Spicaria anomala</i>)
<i>Leptinotarsa decimlineata</i> Say (8, 9, 11)	18. Blue stain of spruce (<i>Ceratostomella piceaperda</i>)
<i>Orsodacne attia</i> (Ahr.) (3)	19. Blue stain of Douglas fir (<i>Ceratostomella pseudotsugae</i>)
<i>Systema taeniata</i> (Say) (8, 9)	20. Tomato leaf spot (<i>Septoria lycopersici</i>)
Dermistidae:	
<i>Attagenus piceus</i> Oliv. (3)	
<i>Anthrenus</i> sp. (3)	
<i>Glischrochilus fasciatus</i> Oliv. (3)	
Curculionidae:	
<i>Cryptorhynchus lapathi</i> L. (12)	
Elateridae:	
<i>Melanotus oregonensis</i> (Le C.) (3)	
Scolytidae:	
<i>Dendroctonus ponderosae</i> Hopk. (13)	
<i>Dendroctonus piceaperda</i> (18)	
<i>Dendroctonus pseudotsugae</i> (19)	
<i>Ips grandicollis</i> Eich. (13)	
<i>Ips interger</i> Eich. (13)	
<i>Ips pini</i> Say (13)	
<i>Ips emarginatus</i> (Le C.) (13)	
<i>Ips oregoni</i> (Eich.) (13)	
<i>Scolytus rugulosus</i> Ratz. (3)	
<i>Scolytus scolytus</i> Fabr. (14)	
<i>Scolytus multistriatus</i> Marsh. (14)	
<i>Scolytus sulcifrons</i> Rey (14)	
<i>Scolytus affinis</i> Eggers (14)	
<i>Scolytus laevis</i> Chapuis (14)	
<i>Scolytus pygmaeus</i> Fabr. (14)	
<i>Scolytus sulcatus</i> Le C. (14)	
<i>Scolytus ventralis</i> Le C. (16)	
<i>Scolytus praeceps</i> Le C. (17)	
<i>Scolytus subscaber</i> Le C. (17)	

TABLE II.—SOME OF THE MORE IMPORTANT VECTORS OF PLANT DISEASES AND THE DISEASES THAT THEY TRANSMIT.—(Continued)

Vectors	Diseases
LEPIDOPTERA	
Pyralididae:	
<i>Mimorista flavidissimalis</i> Grote (1)	1. Bacteriosis of prickly pear plants (<i>Erwinia cacticida</i>)
Phycitidae:	2. Fire blight (<i>Erwinia amylovora</i>)
<i>Cactoblastis bucyrus</i> Dyar (1)	3. Black rot of crucifers (<i>Phytophthora campestri</i>)
<i>Cactoblastis cactorum</i> Berg. (1)	4. Plum wilt (<i>Lasiodyplodia triflorae</i>)
<i>Melitara prodenialis</i> Walker (1)	
<i>Melitara junctelinsella</i> Hulst (1)	
Aegeriidae:	
<i>Aegeria exilosa</i> Say (4)	
Olethreutidae:	
<i>Carpocapsa pomonella</i> L. (2)	
Papilionidae:	
<i>Plusia brassicae</i> Riley (3)	
DIPTERA	
Mycetophilidae:	
<i>Sciara thomae</i> L. (1)	1. Ergot of cereals and grasses (<i>Claviceps paspali</i>)
Bibionidae:	2. Fire blight (<i>Erwinia amylovora</i>)
<i>Bibio albipennis</i> Say (2)	3. Olive knot (<i>Erwinia savastanoi</i>)
Trypetidae:	4. Bacterial rot of apples (<i>Phytophthora melophthora</i>)
<i>Dacus oleae</i> Rossi (3)	5. The heart rot of celery (<i>Erwinia carotovora</i>)
<i>Rhagoletis pomonella</i> Walsh (4)	6. Souring of figs (yeast)
Drosophilidae:	7. Smut of figs (<i>Aspergillus niger</i>)
<i>Drosophila ampelophila</i> Leow. (6, 7)	8. Blackleg of cabbage (<i>Phoma lingam</i>)
<i>Drosophila funebris</i> Fabr. (2)	9. Potato blackleg (<i>Erwinia carotovora</i>)
<i>Elachiptera costata</i> Leow. (5)	10. Soft rot of crucifers (<i>Erwinia carotovora</i>)
<i>Scaptomyza graminum</i> Fall. (5)	
Anthomyiidae:	
<i>Hylemyia antiqua</i> Meig. (2)	
<i>Hylemyia brassicae</i> Bouché (8, 10)	
<i>Hylemyia cilicrura</i> Rond. (9)	
<i>Hylemyia lipsia</i> Walker (2)	
<i>Hylemyia trichodactyla</i> Rond. (9)	
<i>Pegomyia calyptata</i> Zett. (2)	
Calliophoridae:	
<i>Cynomyia cadaverina</i> Desv. (2)	
Muscidae:	
<i>Musca domestica</i> L. (2)	
<i>Muscina assimilis</i> Fall. (2)	
<i>Muscina stabulans</i> Fall. (2)	

TABLE II.—SOME OF THE MORE IMPORTANT VECTORS OF PLANT DISEASES AND THE DISEASES THAT THEY TRANSMIT.—(Continued)

Vectors	Diseases
HYMENOPTERA	
Chalcididae:	
<i>Blastophaga psenes</i> L. (1)	1. Endosepsis of figs (<i>Fusarium moniliforme</i> var. <i>fici</i>)
Formicidae:	2. Fire blight (<i>Erwinia amylovora</i>)
<i>Formica fusca</i> L. var. <i>subserica</i> Say (2)	3. Downy mildew of lima beans (<i>Phytophthora phaseoli</i>)
<i>Formica pallidefulva</i> Latr. subsp. <i>schaufussi</i> Mayr. (2)	
<i>Lasius niger</i> (L.) var. <i>americanus</i> Emerv. (2)	
<i>Prenolepis imparis</i> Say (2)	
Vespidae:	
<i>Polistes</i> sp. (2)	
<i>Vespula</i> sp. (2)	
Apidae:	
Bees (3)	
ACARINA (MITES):	
<i>Eriophyes ribis</i> (Westw.) Nal. (1)	1. Reversion of currants (<i>Ribes virus</i> 1)
<i>Pediculopsis graminum</i> Reut. (2)	2. Silver top of June grass and bud rot of carnation (<i>Sporotrichum poae</i>)
SLUGS:	
<i>Agriolimax agrestis</i> L. (1)	1. Black rot of crucifers (<i>Phytonomonas campestris</i>)
NEMATODES:	
<i>Aphelenchus avenae</i> Bost (6)	1. Dilophospora disease of cereals
<i>Anguillulina dipsaci</i> Kuhn (2)	2. Potato tuber rot
<i>Anguillulina similis</i> (Cobb) Goodey (5)	3. "Potato sickness" (<i>Colletotrichum atromentarium</i>)
<i>Heterodera schachtii</i> (3)	4. Root rot of cereals
<i>Tylenchus multicincta</i> Cob. (6)	5. Root rot of sugar cane
<i>Tylenchus pratensis</i> de Man (4) (6)	6. Soft rot (<i>Erwinia carotovora</i>)
<i>Tylenchus tritici</i> (Steinbuch) Bastian (1)	
BIRDS:	
<i>Certhia familiaris americana</i> (Bonap) (1)	1. Chestnut blight (<i>Endothia parasitica</i>)
<i>Dryobates pubescens medianus</i> (Swainson) (1)	
<i>Junco hyemalis</i> L. (1)	
<i>Mniotilta varia</i> L. (1)	
<i>Regulus satrapa</i> Licht. (1)	
<i>Sitta carolinensis</i> Lath. (1)	
<i>Sphyrapicus varius</i> L. (1)	

TABLE III.—A COMPARISON OF CERTAIN REPRESENTATIVE FUNGUS DISEASES OF PLANTS IN RESPECT TO TRANSMISSION PHENOMENA

Disease	Pathogen	Known insect vectors	Nature of insect transmission	Is vector obligatory in nature?	Does pathogen hibernate in vector?	Congenital transmission in vector?
Ergot of cereals	<i>Claviceps purpurea</i>	Many species of flies	Mechanical; dissemination without wounding	No	No positive evidence	No
Blue stain of conifers	<i>Ceratosomella ips</i> , <i>C. spp.</i> , <i>Tuberulariella ips</i> , and other fungi	<i>Ips pini</i> , <i>I. grandicollis</i> , and other bark beetles	Mechanical; dissemination with wounding	Yes	Yes	No
Dutch elm disease	<i>Ceratosomella ulmi</i>	<i>Scolytus scolytus</i> , <i>S. multistriatus</i> , <i>S. sulcifrons</i> , <i>S. affinis</i> , <i>S. pygmaeus</i> , and <i>Hylurgopinus rufipes</i>	Mechanical; dissemination with wounding	Yes	Yes	No
Endosepsis of figs	<i>Fusarium moniliforme</i> var. <i>ficis</i>	<i>Blastophaga pænes</i>	Mechanical; dissemination without wounding	Yes	No	No
Souring of figs	Two or more unnamed species of yeast	<i>Carpophilus hemipterus</i> , <i>Drosophila ampelophila</i> , and several species of thrips	Mechanical; dissemination with and without wounding	No; but development is largely dependent upon the vectors	No	No
Figsmut.	<i>Aspergillus niger</i>	<i>Carpophilus hemipterus</i> , <i>Drosophila ampelophila</i> , and several species of thrips	Mechanical; dissemination with and without wounding	No; but development is largely dependent upon the vectors	No	No
Stigmatomyces of cotton, beans, citrus, and other plants.	<i>Nematospora</i> spp., <i>Spermophthora gossypii</i> , and <i>Eremothecium cymbalariae</i>	<i>Dysdercus</i> spp., <i>Nezara hilaris</i> , and other plant bugs	Biological?	Yes	Not known	Not known
Perennial canker of apple.	<i>Gleosporium perennans</i>	<i>Schizoneura lanigera</i>	Mechanical; wounding without dissemination	Yes	No	No
Downy mildew of lima beans.	<i>Phytophthora phaseoli</i>	Bees	Mechanical; dissemination without wounding	No	No	No
Tree cricket canker of apple and raspberry.	<i>Leptosphaeria coniothyrium</i>	<i>Oecanthus niscus</i> , <i>O. angustipennis</i>	Mechanical dissemination with wounding	No	No	No

Dwarf disease of rice.	No	Not known	No	No	<i>Nephotettix epirata</i> var. <i>chitrensis</i> and <i>Deltocephalus dorsalis</i>	Yes	An incubation period required but minimum time not established
Spotted wilt....	Yes	3 hr.	No	No	<i>Tripsa tabaci</i> and <i>Frankliniella trypocetes</i>	No	Adults cannot acquire the virus by feeding. They may transmit the virus but must acquire it in the larval stage	5 to 7 days
Yellow spot of pineapple.	Yes, but with difficulty	Not known	Not known	Not known	<i>Tripsa tabaci</i>	No	Adults cannot acquire the virus by feeding. They may transmit the virus but must acquire it in the larval stage
Crinkle leaf of sugarcane.	Yes, with difficulty; 13% of trials	Not known	No	No	<i>Piruma quadrata</i> Febb.	No	Transmitted by adults only	Variable; not definitely known
Leaf curl of cotton	No	Not known	No	No	<i>Bemisia gasariparva</i>	No	Not more than 30 min.
Latent (X) virus of potato.	Yes	6 weeks or longer	No	No	None
Tobacco mosaic..	Yes, readily	3 months to 3 years or more; very resistant	No	No	<i>Mycia persica</i> , <i>M. parvioralis</i> , <i>Myndus plagiator</i> , with difficulty and to certain hosts only	No	This virus has been isolated in the form of large prismatic crystals and many physical and chemical properties determined	None
Wheat mosaic...	Yes	Not known	No	No	None known	Infection in nature only under certain conditions through either roots or crown. Aerial spread absent

TABLE V.—A COMPARISON OF CERTAIN REPRESENTATIVE BACTERIAL DISEASES OF PLANTS IN RESPECT TO TRANSMISSION PHENOMENA

Disease	Pathogen	Known insect vectors	Nature of insect transmission	Is vector obligatory in nature?	Does pathogen hibernate in vector?	Congenital transmission in vector?
Fire blight	<i>Eruinia amylovora</i>	Bees, wasps, flies, and many sucking insects	Mechanical; dissemination with and without wounding	No	No	No
Cucurbit wilt	<i>Eruinia tracheiphila</i>	<i>Diabrotica vittata</i> and <i>D. 12 punctata</i>	Biological; dissemination with wounding	Yes	Yes	No
Soft rot of plants	<i>Eruinia carotovora</i>	<i>Hylemyia cilicrura</i> , <i>H. brassicae</i> , and other dipterous insects	Biological; dissemination with wounding	No	Yes	No
Olive knot	<i>Eruinia savastanoi</i>	<i>Dacus oleae</i>	Biological; dissemination with wounding	No	Yes	Yes
Wilt of corn	<i>Phytomonas stewarti</i>	<i>Chaetocnema pulicaria</i> , <i>C. denticulata</i> , <i>Diabrotica longicornis</i> , <i>Hylemyia cilicrura</i>	Biological; dissemination with wounding	No; but development is largely dependent upon the vectors	Yes	No evidence
Apple rot	<i>Phytomonas melophthora</i>	<i>Rhagoletis pomonella</i>	Biological; dissemination with wounding	Yes	No positive evidence	No

TABLE VI.—A COMPARISON OF CERTAIN REPRESENTATIVE DISEASES OF ANIMALS IN RESPECT TO TRANSMISSION PHENOMENA

Disease	Pathogen	Athropod vector	Definitive host	Intermediate host	Method of transmission	Incubation period in vector	Congenital transmission in vector?
Filariasis (elephantiasis).	<i>Filaria bancrofti</i>	Several species of mosquito	Man	Mosquitoes, several different species	Contaminative, following bite of infective mosquito	16 to 25 days	No
Texas fever.....	<i>Babesia bigema</i>	Cattle ticks; <i>Borophagus annulatus</i> , <i>B. spp.</i> , and <i>Ixodes ricinus</i>	Cattle ticks, <i>Borophagus annulatus</i> , <i>B. spp.</i> , and <i>Ixodes ricinus</i>	Cattle	Inoculative, by bite of the ticks	One complete generation of the vector	Yes; the disease is transmitted only by ticks that feed on infected cattle
Rocky Mountain spotted fever.	<i>Dermacentor variabilis</i>	The tick (<i>Dermacentor variabilis</i>)	?	?	Inoculative, by bite of infective tick (<i>Dermacentor variabilis</i>)	At least 9 days	Yes
The nagana disease of cattle.	<i>Trypanosoma brucei</i>	The tsetse fly (<i>Glossina morsitans</i>)	The tsetse fly (<i>Glossina morsitans</i>)	Cattle	Inoculative, by bite of the tsetse fly	10 days	No
Sleeping sickness of man.	<i>Trypanosoma gambiense</i>	The tsetse flies (<i>Glossina palpalis</i> and <i>G. tachinoides</i>)	The tsetse flies (<i>Glossina palpalis</i> and <i>G. tachinoides</i>)	Man	Inoculative, by bite of the tsetse flies	20-30 days	No
Malaria.....	<i>Plasmodium vivax</i> , <i>P. malariae</i> , and <i>P. falciparum</i>	The anophelinae mosquitoes	The anophelinae mosquitoes	Man	Inoculative, by bite of the mosquito	8-14 days	No
Yellow fever.....	A virus	<i>Aedes acgypti</i> and several other species of mosquito	?	Man and monkeys	Inoculative, by bite of the mosquito	9-14 days	No
Dengue.....	A virus	<i>Aedes acgypti</i> , <i>A. albopictus</i> , and other mosquitoes	?	Man and monkeys	Inoculative, by bite of the mosquito	8-11 days	No
Bubonic plague.....	<i>Pasteurella pestis</i>	<i>Xenopsylla cheopis</i> and other species of insects	Inoculative, by bite of flea, and in excreta of flea	None	No
Typhus fever.....	<i>Rickettsia prowazekii</i>	The body louse (<i>Phedusa humanus</i>)	?	?	Inoculative, by bite of louse or contaminative in excreta of louse	8-9 days	No
Trench fever.....	<i>Rickettsia quintana</i>	The body louse (<i>Phedusa humanus</i>)	?	?	Inoculative, by bite of louse or contaminative in excreta of louse	5-9 days	No
Typhoid fever.....	<i>Escherichia typhosa</i>	<i>Macra domestica</i> and related species	Contaminative, both internally and externally	None	No

GLOSSARY

The author appreciates the difficulty of accurately defining any biological concept. Definitions, however, do aid in straight thinking, and failure to define terms often leads to confusion. Phytopathology and entomology are young sciences, and their terminologies are in the process of evolution. Consequently, so many of the terms have been used so loosely that some degree of definition is necessary if confusion is to be avoided.

Moreover, the sciences of phytopathology and entomology have developed their own distinctive terminologies which are not always clearly understood by specialists in the other field. The definitions given here are not intended to be invulnerable, and some people may object to the sense in which certain terms have been used. The definitions are included in the belief that they may be of assistance to some of the readers of this book.

Anemophilous. Wind-pollinated.

Antagonistic symbiosis. Symbiosis in which one member of the association benefits at the expense of the other. Parasitism.

Antibiosis. An association between two or more organisms that is detrimental to one or more of them.

Arthropod. An organism with annulate body and segmented appendages, of Phylum Arthropoda which includes insects, mites, spiders, crustacea, centipedes, etc.

Ascospores. Sexually produced fungus spores borne in a special cell known as an "ascus."

Autocatalytic. Stimulating a chemical reaction that results in the production of more of the stimulating substance.

Autogenetic. Pertaining to the spontaneous origin of living organisms.

Basement membrane. A thin noncellular layer constituting the internal limits of the epidermis of insects.

Basipetally. Successively from the apex to the base.

Bromatia. The peculiar enlarged ends of the mycelium of fungi cultivated by ants.

Buccal cavity. The foremost part of the stomodaeum lying just inside the mouth.

Bursa copulatrix. A modification of the vagina serving as a copulatory pouch.

Caecum (*pl.* caeca). An evaginated blind sac opening into the intestinal tract.

Callus. Soft parenchymatous tissue of cambial origin formed over a wound.

Carrier. In medicine, a person who is a constant host for an infectious pathogen but manifests no clinical symptoms of the disease. In plant pathology, a plant that contains a virus but develops no symptoms of a

virus disease. In earlier literature, "carrier" was used to designate an insect that transmitted a virus, but the term "vector" is now preferred.

Cecidium. A plant gall.

Chlamydospore. A thick-walled spore developed directly from hyphal cells and not borne on special sporophores.

Chlorosis. A diseased condition characterized by a deficiency of chlorophyll.

Chlorotic. Possessing less than the normal amount of chlorophyll.

Chorion. The egg shell.

Close pollination. The transference of pollen from the stamen of one flower to the stigma of another flower of the same plant.

Clypeus. The facial area of the cranium of insects lying just above the labrum.

Colleterial glands. Female accessory glands that secrete an adhesive substance used to fasten eggs to some sort of support.

Colon. The posterior part of the hind-intestine, between the ileum and the rectum.

Commensalism. Symbiosis in which neither party is injured or benefited or in which one member of the association is benefited without either benefit or injury to the other.

Congenital transmission. Transmission from one generation to the next successive generation through the process of reproduction.

Conidium. A fungus spore produced asexually and exogenously.

Contagious. Spread by contact.

Contaminative. Being transmitted by surface contamination without wounding by the vector.

Coprophilous. Growing on the dung of animals.

Coremia. A fruiting structure of fungi consisting of a group of erect intertwined hyphae bearing conidia at their apex.

Crop. An enlargement of the esophageal region of the stomodaeum, often serving as food reservoir.

Cross-pollination. The transference of pollen from the anther of flowers on one plant to the stigma of flowers on another plant.

Cuticle. A layer of cutin found on the outside of the outer walls of epidermal cells of plants.

Cuticula. The outer noncellular layer of the body walls of insects.

Cutin. A tough waxy substance secreted on the surface of epidermal cells of plants.

Cyclical. Involving a sexual reproduction cycle.

Definitive host. The host in which the sexual stage of a parasite is found.

Diploidization. The bringing together, in the same mycelium, of nuclei of opposite sex.

Dissemination. To scatter or diffuse. The transportation of inoculum from one location to another. Does not imply either inoculation, ingression, or infection (*cf.* Transmission).

Diverticulum. A blind saclike offshoot from the alimentary canal or other organs.

Ecdysis. Molting; the process of casting off the skin.

- Ectosymbiosis.** Symbiosis in which the microsymbiote develops on the outside of the body of the other symbiote.
- Endemic.** The regular occurrence of a disease of man with little variation in abundance.
- Endocuticula.** The inner layer of the cuticula; usually not heavily sclerotized.
- Endogenous.** Arising from within the generating structure.
- Endophylaxis.** Plant resistance to insects caused by internal physiological properties.
- Endosymbiosis.** Symbiosis in which the microsymbiote develops within the body of the other symbiote.
- Enphytotic.** The regular occurrence of a disease of plants in a given locality with little variation in abundance. Analogous to, but not synonymous with, endemic.
- Enteric.** Relating to the intestinal organs.
- Entomophily.** Pollination of flowering plants through the agency of insects.
- Enzootic.** The regular occurrence of a disease of animals with little variation in abundance.
- Epicuticula.** A very thin nonchitinous layer on the outer surface of the cuticula.
- Epidemic.** The occurrence of a disease of human beings in abundant and destructive proportions.
- Epidemiology.** The study of epidemics.
- Epidermis.** The outermost layer of cells on most plant organs before the development of periderm.
- Epipharynx.** One of the mouth parts of insects, attached to the inner surface of the labrum; probably an organ of taste.
- Epiphyllaxis.** Plant resistance to insects caused by external morphological factors.
- Epiphyte.** A plant that lives upon another plant but derives no nourishment from it. A place parasite.
- Epiphytotic.** The occurrence of a disease of plants in abundant and destructive proportions. Analogous to, but not synonymous with, epidemic.
- Epithelium.** A layer of cells covering the surface or lining a cavity.
- Epizootic.** The occurrence of a disease of animals in abundant and destructive proportions.
- Esophagus.** The tubular portion of the stomodaeum between the pharynx and the crop.
- Evagination.** The protrusion of an inner surface toward the outside.
- Exfoliated.** Separated in a thin layer from the underlying tissue.
- Exocuticula.** The outer layer of the cuticula; the layer most commonly sclerotized.
- Exogenous.** Arising on the outside of the generating structure.
- Facultative parasite.** An organism normally living as a saprophyte but that can live as a parasite.
- Facultative saprophyte.** An organism normally living as a parasite but that can live as a saprophyte.

- Filter chamber.** A part of the alimentary canal in Homoptera in which the anterior end of the mesenteron and the beginning of the hind-intestine are bound together, providing a means for water and carbohydrate solutes to pass by diffusion into the hind-intestine without passing through the mesenteron.
- Flagellate.** Possessing one or more flagella; a protozoan belonging to the Mastigophora and characterized by having one or more flagella.
- Flagellosis.** A disease caused by flagellate protozoa.
- Follicle.** Egg chamber; a compartment of the egg tube that contains an oöcyte.
- Galeae.** The outer lobes of the maxillae.
- Germarium.** The end chamber of an ovariole (or testicular tube) containing the primary oögonium (or spermatogonia).
- Glabrous.** Having few or no trichomes.
- Glossae.** The two median lobes of the labium.
- Gravid.** Pregnant; ready to bear young.
- Guttation.** Loss of water by exudation through hydathodes.
- Hemolymph.** The clear watery liquid in the body cavity of insects and other invertebrates. The "blood" of insects.
- Heterothallism.** That condition in which the fusion of two mycelia of different nuclear complement is necessary for sexual reproduction.
- Hibernation.** The act of surviving the winter usually in a dormant or quiescent condition.
- Homosexual.** Producing only one kind of gamete.
- Host.** An organism on or in which a parasite lives.
- Hydathodes.** Openings through the epidermis of leaves usually located near the terminal veinlets. They have no guard cells and serve for water loss by guttation when relative humidity is high.
- Hymenium.** The fruiting surface of Ascomycetes or Basidiomycetes.
- Hyperplasia.** Overgrowth caused by increase in number of cells.
- Hypertrophy.** Overgrowth caused by increase in cell size.
- Hypodermis.** The epidermis of insects; the cell layer of the body wall of insects.
- Hypopharynx.** A tongue-like prolongation of the floor of the mouth of insects, attached to the inside wall of the labium.
- Hypopus.** A stage in the development of certain mites especially adapted for their dissemination by insects to whose bodies they cling by means of special organs of suction.
- Ileum.** The anterior part of the hind-intestine between the mesenteron and the colon.
- Imperfect stage (of a fungus).** The stage that does not include spores formed as a result of a sexual process.
- Incubation period.** That time elapsing between the time of inoculation and infection (in reference to a plant pathogen). In general, the period of embryonic development. The time elapsing between ingestion of an infectious agent by an insect and the time when the insect becomes infective.

- Infection.** That process in the development of a disease in which the pathogen becomes established on the suscept and begins to drive its nourishment from it.
- Infection court.** That portion of a suscept through which a pathogen may infect under suitable environmental conditions.
- Infectious.** Capable of being transmitted by inoculation.
- Infective.** Able to communicate infection. An insect is said to be "infective" when, after ingesting an infectious agent, it is able to induce infection by feeding on the suscept. The same as "viruliferous" when used in connection with virus diseases.
- Infestation.** To be present in large quantities in a given medium or locality.
- Infrabuccal pouch.** In ants, a spherical sac below the floor of the mouth and opening into the mouth.
- Ingression.** The act of gaining entrance into the tissues of the suscept. Infection may or may not follow ingression. Ingression may be accomplished by some pathogens without external aid. Others require the aid of some wound-producing agent.
- Inoculation.** The act of placing inoculum on the infection court (or an assumed infection court). In medical science, the term usually implies insertion under the skin, thus including also the act of ingression, which is excluded in the sense used here.
- Inoculative.** Being transmitted through injection under the skin as by an insect vector.
- Inoculum.** Infective material; a pathogen or part of a pathogen that may infect and cause a disease.
- Instar.** The period or stage between molts in the metamorphosis of insects.
- Intermediate host reservoir.** Hosts in which a supply of the asexual stage of a parasite occurs.
- Invagination.** The retraction of a outer surface toward the inside.
- Invasion.** The spread of a pathogen through the tissues of a suscept.
- Isogamic.** Sexual reproduction by morphologically similar gametes.
- Kataplastic.** Overgrowths in which the new tissue is composed of undifferentiated elements.
- Klendusic.** Disease-escaping; having the property of klendusity.
- Klendusity.** The property of escaping infection under natural conditions although susceptible when inoculated artificially.
- Labella.** The sensitive spongelike organs at the end of the proboscis of certain Diptera; formed by a modification of the tip of the labium.
- Labium.** The lower tip of the insect mouth parts formed by the union of a second pair of maxillae.
- Labrum.** The upper lip of insects, covering the base of the mandibles and forming the roof of the mouth.
- Latent period.** The time elapsing between inoculation and the appearance of symptoms of the disease.
- Latex.** A viscid milky juice secreted by specialized cells of certain plants.
- Laticiferous.** Producing latex.
- Lesion.** A morphological or histological symptom of a disease.

Longevity. Ability to live or survive for a long time.

Macrogametocyte. The female sex cell arising from the merozoite of the malarial protozoan.

Malpighian tubules. Long slender tubules opening into the alimentary tract of insects at the anterior end of the hind-intestine and having excretory functions.

Mandibles. The first pair of appendages composing the mouth parts of insects. They assume various forms in different groups of insects.

Maxillae (the first maxillae). The second pair of appendages of the mouth parts of insects.

Meristematic. Capable of producing new tissue by cell division.

Merozoite. The third stage in the asexual development of the protozoan causing malaria.

Mesenteron. The middle portion of the alimentary canal of insects which is of entodermal origin; the mid-intestine.

Mesophyll. The chlorophyll parenchyma of leaves.

Metamorphosis. Change of form; the progressive changes of form of an insect in growing from the egg through larva, pupa, to adult.

Microgametocyte. The male sex cell arising from the merozoite of the malarial protozoan.

Micropyle. A minute opening in the insect egg through which the spermatozoa enter in fertilization.

Microsymbiote. A microorganism that lives in symbiosis with a more highly developed organism.

Microtrichia. Nonarticulated hairs formed as extensions of the cuticula of insects.

Mimetic. Imitative.

Monophagous. Feeding on one plant or kind of food.

Mutualism. Mutualistic symbiosis.

Mutualistic symbiosis. Symbiosis that is beneficial to both of the members of the association.

Mycetocyte. A cell that harbors symbiotic microorganisms.

Mycetome. A tissue composed of mycetocytes.

Mycophagous. Feeding upon fungi.

Necrosis. The death and destruction of cells or tissues.

Necrotic. Characterized by rapid death and destruction of tissue.

Oligophagous. Feeding on a limited number of plants or kinds of food.

Oöcyst. A resting nodular stage of the malarial parasite formed from the oökinete.

Oöcyte. The differentiated egg cell just before maturation.

Oögonium. The first stage in the differentiation of the egg cell from the primary germ cell.

Oökinete. The zygote of the malarial parasite.

Ovaries. The mass of ovarian tubules in which the eggs are developed.

Ovarioles. The egg tubes that together make up the ovary.

Oviduct. One of the paired tubes through which the egg passes from the ovarian tubes into the vagina.

Oviparous. Reproducing by eggs.

- Ovipositor.** The tubular organ of female insects by means of which eggs are deposited.
- Ovoviviparous.** Producing living young by the hatching of the egg while still in the body of the mother.
- Parasite.** An organism living in or on another living organism of a different species from which it derives all or part of its nourishment.
- Parasitism.** Living as a parasite.
- Parenchyma.** The thin-walled isodiametric cells of plant tissue.
- Parthenogenesis.** Reproduction by direct growth of egg cells without fertilization by the male element.
- Pathogen.** An organism that can cause a disease.
- Pathogenicity.** The ability of an organism to cause disease.
- Perfect stage** (of a fungus). The stage that includes the spores formed as a result of a sexual process.
- Periderm.** A layer of cork cells on the outside of plant roots and stems and formed by growth of a cork cambium.
- Pharynx.** The back part of the mouth and the fore part of the esophagus.
- Physiologic race.** A biotype of group of closely related biotypes differing from other biotypes in physiological behavior.
- Phytoecidium.** A plant gall caused by the action of a plant.
- Phytophagous.** Feeding on plants.
- Phytotoxic.** Toxic to plants.
- Plasmodesma.** Delicate threads of protoplasm that pass through the walls between two cells, uniting the protoplasts of the two cells.
- Pleomorphism.** The inclusion of many different forms in the normal life cycle.
- Pollination.** The transference of pollen from the stamen to the stigma of flowers.
- Pollinia.** Masses of sticky pollen grains borne in packets and especially adapted to transportation by insects.
- Polyphagous.** Feeding on many different plants or kinds of food.
- Preservation.** Insurance of the survival of a pathogen by protecting it from unfavorable environmental conditions.
- Prestomal teeth.** Toothlike structures arising from the cleft between the labella lobes of the proboscis of certain Diptera.
- Primary infection.** First infection following a period of quiescence of a pathogen.
- Proboscis.** The extended or prolonged mouth parts of an insect.
- Proctodaeum.** The posterior part of the alimentary canal of insects which is of ectodermal origin; the hind-intestine.
- Prosoplastic.** Overgrowth in which new tissue is composed of well-differentiated elements.
- Protoplasmic membrane.** A semipermeable membrane consisting of the thin outer layer of the protoplasm of a cell.
- Protozoa.** Single-celled animals without true tissues or true organs.
- Proventriculus.** A specialized part of the fore-intestine immediately anterior to the ventriculus.
- Pseudotracheae.** False tracheae.

- Pseudovitellus.** The mycetome of aphids.
- Pubescent.** Having abundant trichomes.
- Pycnidium.** A hollow multicellular fruiting body in which asexual spores are produced; characteristic of the Sphaeropsidales and certain Ascomycetes.
- Pycnium.** A pycnidiumlike structure in the rusts in which are produced haploid spores that serve as agents of diploidization.
- Rectum.** The posterior part of the hind-intestine, opening at the anus.
- Rickettsia.** Minute bacteriallike microorganisms that live symbiotically in the bodies of insects by which they often may be transmitted to higher animals on which they may become pathogenic.
- Roguing.** The removal of plants because of disease or other undesired characters.
- Saprophyte.** A plant that lives upon dead organic matter.
- Sclerotized.** Infiltrated with a hard or tough substance.
- Secondary infection.** Infection in a period of active spread of a disease.
- Self-pollination.** The transference of pollen from the stamen to the stigma of the same flower.
- Setae.** Hollow, pointed, articulated hairs formed by a modified epidermal cell.
- Setal sheath.** A sheath surrounding the feeding puncture made in plant tissue by a homopterous insect; probably formed by the saliva of the insect reacting with the plant sap.
- Spermatheca.** The reservoir in the female insect which receives the sperm during or shortly after copulation.
- Sporidium.** Small spores produced on the basidia or promycelia of smuts and rusts.
- Sporozoites.** Minute individuals produced by the oöcyst of the malarial parasite. The form that invades the salivary glands of the mosquito.
- Stele.** The axis or central cylinder of vascular plants consisting of the conducting tissues and associated supporting tissues.
- Stigma.** The surface of the style of a flower through which the germinating pollen grain penetrates to reach the ovary.
- Stigmonose.** A diseased condition resulting from localized injury caused by insect punctures.
- Stomata** (*sing.* stoma). Specialized openings in the epidermis adapted for exchange of gases.
- Stomodaeum.** The anterior part of the alimentary canal of insects which is of ectodermal origin; the fore-intestine.
- Stylets.** Small slender sclerotized processes of the insect exoskeleton.
- Suscept.** An organism that is susceptible to a given disease.
- Symbiote = Symbiont.** One partner of a symbiotic relationship. Commonly applied to a microorganism symbiotic with a more highly developed organism.
- Symbiosis.** The condition of living together of two organisms of different species in close spatial relationship. Often used in a restricted sense implying mutualistic symbiosis.
- Teliospore.** A spore of the Uredinales, usually a resting spore, that on germination produces a promycelium or a basidium.

Thorax. The second or intermediate region of the insect body, bearing the true legs and wings.

Toxicogenic. Capable of producing a toxic substance. Insects that introduce a toxic substance into the plant while feeding are said to be "toxicogenic."

Toxiniferous. Actively producing a toxic substance. Toxicogenic insects may temporarily lose their ability to produce the toxic substance, in which case they would not be toxiniferous.

Transmission. The acts of disseminating and inoculating inclusively; the act of perpetuating a disease from one generation to another as through seed or vegetative propagation.

There is some difference of opinion among plant pathologists in regard to the use of the phrase "disease transmission." Some maintain that a disease, not being a concrete body, cannot be transmitted, only the pathogen or infectious agent being subject to transmission. The same objection is raised to the use of "disease dissemination." The author believes that these objections are not entirely valid and that there is ample justification, both etymological and traditional, for the use of either phrase. However, from the standpoint of the phenomena of plant disease development, there appears to be some justification for not using the two terms interchangeably. In this book, an effort has been made to use the term "dissemination" only in connection with the pathogen. This restricted use is based on the concept that the pathogen may be *disseminated* widely but the disease is not *transmitted* until inoculation and infection have occurred. Thus, an insect may *disseminate* a pathogen without *transmitting* the disease.

Transpiration. Loss of water by evaporation through stomata.

Trichogen. The modified epidermal cell of insects from which the setae arise.

Trichomes. Modified hairlike epidermal cells of plants.

Vagina. The egg passage formed by the union of the oviducts and through which the egg passes to the ovipositor.

Vector. An agent of dissemination or inoculation or both.

Ventriculus. The mesenteron, or mid-intestine.

Viable. Living or capable of growth.

Virulence. The degree of ability to produce disease. A measure of pathogenicity.

Viruliferous. Capable of inducing a virus disease by feeding on the susceptible. Infective, in regard to virus diseases.

Virus. An ultramicroscopic, filterable, infectious agent.

Vitellarium. That part of the egg tube in which the oöcytes grow to mature size.

Wound cork. Cork tissue formed in the process of healing a wound.

Zooecidium. A plant gall caused by the action of an animal.

Zygote. An individual formed by the union of two gametes of opposite sex.

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